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THE EUSTACHIAN TUBE

A REVIEW OF ITS DESCRIPTIVE, MICROSCOPIC, TOPOGRAPHIC AND CLINICAL ANATOMY

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COLUMBUS, OHIO

A comprehensive survey of the anatomy of the eustachian tube seems timely in view of its importance in relation to some of the problems of aviation and submarine medicine.

Bartholomaeus Eustachius (1520-1574) is accredited with its first description.[1] However, according to Singer,[2] Alcmaeon (about 500 B. C.) "discovered the tubes, called in after years by the name of Eustachius." This author also points out that Aristotle (384-322 B. C.) refers to it in his writings and that "there is, moreover, evidence that two contemporaries of Eustachius, Vesalius (1514-1546) and Ingrassias (1510-1580), were acquainted with it."

Most of the literature dealing with the eustachian tube in modern times is concerned primarily with its function and particularly with the controversy pertaining to whether it is normally open or closed. For a review of the literature bearing on this and related questions the reader is referred to Rich[3] and Perlman.[4]

The objective of the present study of the eustachian tube is a detailed review of its gross and microscopic structure in the adult and the newborn, with emphasis on its location and relations (surface or topographic anatomy) and the related clinical applications, particularly in aviation and submarine medicine and in caisson work.

The method of approach employed was dissection combined with study of celloidin sections made through appropriate levels in such a manner as to illustrate the size, the shape, the location and the relations of the tube. The sections were cut at 40 microns and stained with Masson's stain. In addition, histologic preparations of tubes cut through various planes were used. These tubes were from newborn and adult subjects.

This investigation was aided by a grant from the graduate school to the departments of anatomy and medicine of the Ohio State University

1. Galante, M., and Graves, G. O.: Epistle on the Organs of Hearing: A Translation of Eustachius. Bartholomaeus: Opuscula anatomica—De auditus organis, ed. 2, Lugd. Bat., J. vander Linden, 1707, pp. 124-142, Arch. Otolaryng., to be published.

2. Singer, C.: The Evolution of Anatomy, London, Kegan Paul, Trench, Trubner & Co., Ltd., 1925.

3. Rich, A. R.: A Physiological Study of the Eustachian Tube and Its Related Muscles, Bull. Johns Hopkins Hosp. **31**:206-214 (June) 1920.

4. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

ANATOMY AND PHYSIOLOGY OF THE EUSTACHIAN TUBE OF THE ADULT

The eustachian tube, also designated as the pharyngotympanic or the auditory tube, extends posteriorly, laterally and superiorly from its pharyngeal orifice on the lateral wall of the nasopharynx to its tympanic orifice on the anterior wall of the tympanic cavity (fig. 1). The entire tube consists of two portions, the cartilaginous, which is the anteromedial two thirds, and the osseous, which is the posterolateral one third. The following description of these portions is a clarification based on our own studies of the details collected from various sources.

MEASUREMENTS

The entire tube varies in length from 31 to 38 mm. (1¼ to 1½ inches); the cartilaginous portion measures 24 or 25 mm. and the osseous portion 11 or 12 mm. The usual description states that the tube inclines superiorly at an angle of 30 to 40 degrees with the horizontal plane (the pharyngeal orifice being about 15 mm. [⅗ of an inch] lower than the tympanic orifice). This is based on the assumption that the tube is a straight line from orifice to orifice and not an S-shaped tube as indicated in figure 2. The tube passes laterally from a sagittal plane through the pharyngeal orifice, at an angle of about 45 degrees. According to Piersol,[5] the tube forms an angle of 135 to 145 degrees with the longitudinal axis of the external auditory meatus. This implies that the tube is a straight line, although even Piersol himself stated that the cartilaginous and osseous portions join at a very

5. Piersol, G. M.: Human Anatomy, ed. 9, Philadelphia, J. B. Lippincott Company, 1930.

obtuse angle, which Cunningham [6] mentioned as 160 degrees.

The foregoing measurements were obtained from fixed anatomic material. From living material, Spielberg,[7] by roentgenograms of tubes into which iodized poppyseed oil 40 per cent had been injected, obtained figures similar to the foregoing ones. He stated that the length of the tube is 30 to 33 mm., that the tympanic orifice of the tube is from 2 to 2.5 cm. higher than the

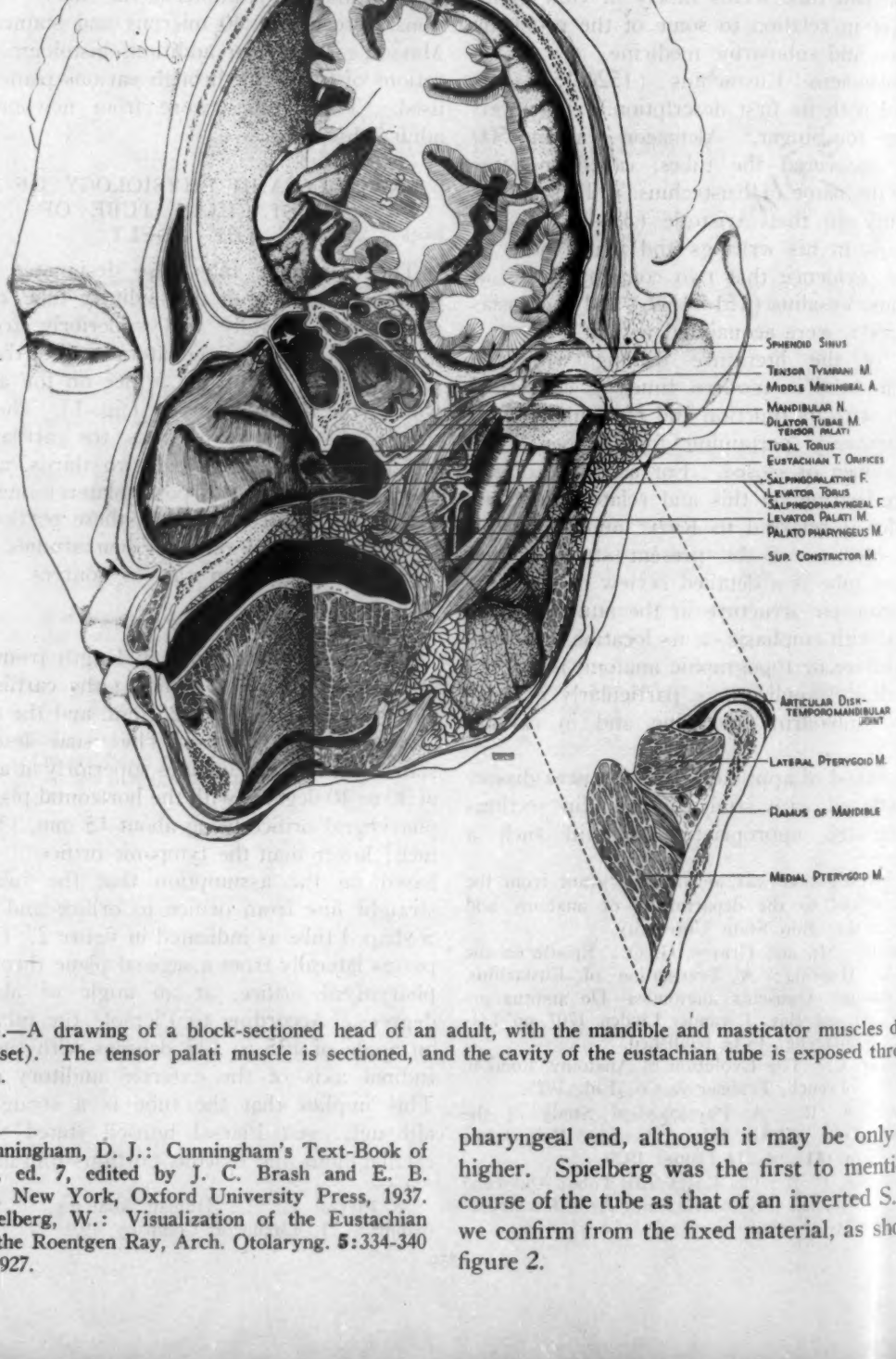


Fig. 1.—A drawing of a block-sectioned head of an adult, with the mandible and masticator muscles dissected away (inset). The tensor palati muscle is sectioned, and the cavity of the eustachian tube is exposed throughout its length.

pharyngeal end, although it may be only 1 cm. higher. Spielberg was the first to mention the course of the tube as that of an inverted S. This we confirm from the fixed material, as shown in figure 2.

6. Cunningham, D. J.: Cunningham's Text-Book of Anatomy, ed. 7, edited by J. C. Brash and E. B. Jamieson, New York, Oxford University Press, 1937.

7. Spielberg, W.: Visualization of the Eustachian Tube by the Roentgen Ray, Arch. Otolaryng. **5**:334-340 (April) 1927.

THE PHARYNGEAL ORIFICE

Nasopharyngoscopic Appearance.—All standard textbooks of anatomy describe the fixed pharyngeal orifice as being either oval or triangular and funnel shaped. On nasopharyngoscopic examination of the living subject the orifice is described as a vertical slit when at rest,[8] which becomes triangular only during the act of swallowing or during the act of raising the soft palate.

Joachim[9] directly observed the orifice of the eustachian tube in a man whose nose had been destroyed on one side by syphilis, including the septum and turbinate bones—the hard and soft palate and the pharynx being left intact. He reported that during rest the lips of the orifice of the tube were in contact in their whole extent and that the cleft was turned forward. When the palate was elevated by sounding o, the prominence of the levator palati muscle pushed itself between the two labia, whereby principally the posterior labium moved back and up. He could perceive only a minimum forward motion in the lowest part of the anterior labium and this not before the elevation of the ridge of the levator palati. The developing prominence of the levator palati did not completely block the orifice of the tube, thus leaving a circular opening between the walls of the latter.

Rich[3] in his nasopharyngoscopic studies of human subjects emphasized that there are differences in the appearance of the orifices among persons, as well as in the same person at different times.

The vertical diameter of the orifice in fixed material varies from 3 to 4 mm.[10] to 8 to 10 mm.[11] and its horizontal diameter from 2 mm.[10] to 5 mm.[12] No measurements on living material have as yet been recorded.

Location and Catheterization.—The site of the orifice must be located in catheterization maneuvers; therefore its variability is to be scrutinized. Urbantschitsch's[13] measurements indicate that the distance from the anterior nasal spine to the pharyngeal orifice is 5.3 cm. to 7.5 cm. More recent measurements made by Mangiaracina[14] on the living subject by means of the nasopharyngoscope indicate somewhat longer dis-

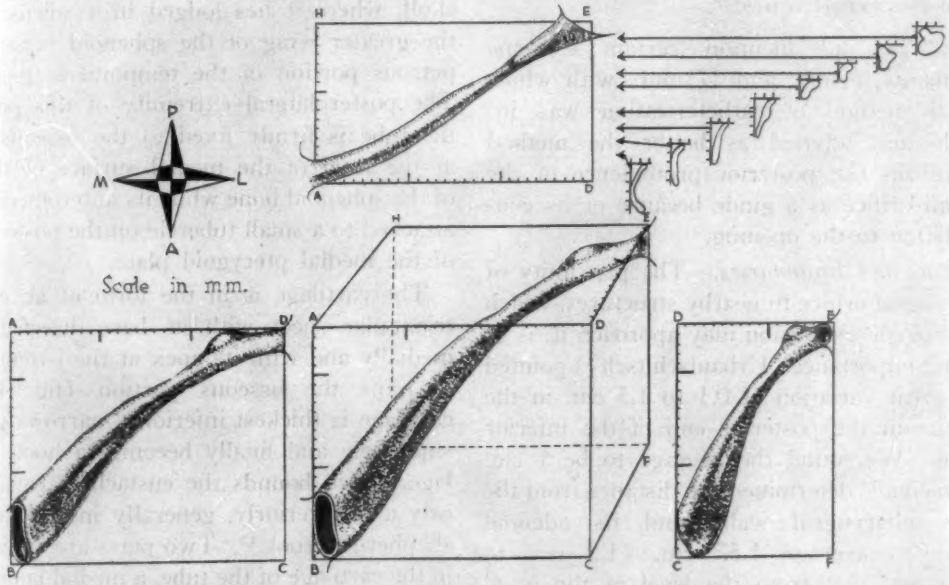


Fig. 2.—The eustachian tube of an adult drawn to scale in its plane and three-dimensional views. The cross-sectional outlines of the tubal lumens are related to the cephalic view *A H E D*. *B A D C* is the anterior view; *C D E F*, the lateral view.

8. Bryant, W. S.: The Eustachian Tube: Its Anatomy and Its Movements, with a Description of the Cartilages, Muscles, Fascia, and the Fossa of Rosenmüller, M. Rec. **71**:931-934 (June 8) 1907.

9. Joachim, O.: Contribution to the Physiology of the Soft Palate, Arch. Otol. **18**:226-234, 1889.

10. Huschke, cited by Urbantschitsch.[13]

11. Quain, J.: Quain's Elements of Anatomy, ed. 11, edited by E. A. Schäfer, J. Symington and T. H. Bryce, London, Longmans, Green & Co., 1909, vol. 3.

12. von Tröltsch, cited by Urbantschitsch.[13]

13. Urbantschitsch, V.: Lehrbuch der Ohrenheilkunde, ed. 2, Leipzig, Urban & Schwarzenberg, 1884, pp. 154-160.

14. Mangiaracina, A.: The Eustachian Orifice (Relation to Landmarks in the Nose and Nasopharynx), Arch. Otolaryng. **35**:649-651 (April) 1942.

tances, from 6.9 to 8.8 cm., with an average of 7.9 cm. He also observed the distance from the posterior edge of the nasal septum to the orifice to vary from 10 to 13 mm. (average, 11 mm.) and gave credence to the usual method of catheterization by saying:

> In introducing and then rotating a catheter having a curvature of 145 degrees and a beak of 20 mm. and then withdrawing it until it strikes the posterior edge of the septum, the beak of the catheter is found to project about 12 mm. into the nasopharynx. When the catheter is then rotated outward 180 degrees it will be opposite the eustachian opening only in those cases in which the distance of the orifice from the posterior edge of the septum is approximately that of the projecting tip of the catheter, viz., about 10 to 12 mm. As in the greater number of cases the distance is between 10 and 13 mm., one is usually successful in entering the tube when this method is used.

However, he did mention certain extreme measurements, 6 mm. and 17 mm., with which the usual method of catheterization was inapplicable and advised as better the method which utilizes the posterior prominence of the eustachian orifice as a guide because of its constant relation to the opening.

Relations and Boundaries.—The proximity of the pharyngeal orifice to nearby structures, which with pathologic expansion may approach it, is of prognostic importance. Urbantschitsch [13] pointed out the great variation of 0.1 to 1.5 cm. in the distance from the posterior end of the inferior turbinate. We found the average to be 1 cm. Mangiaracina [14] determined the distance from the posterior pharyngeal wall (and its adenoid growths) to average 1.57 cm. (1.2 cm. to 2.0 cm.) and that from the level of the nasal floor to average 0.836 cm. (0.5 cm. to 1.2 cm.). Our own figures indicate that the distance from the vault of the nasopharynx averages 1 cm. The distance of the orifice from the surface of the skin in the frontal plane of the temporomandibular joint is 4.5 cm.[15]

The boundaries of the orifice consist of folds anteriorly and posteriorly and elevations superiorly and inferiorly. The posterosuperior boundary of the orifice is most prominent. It is called the tubal elevation (torus tubarius, fig. 1), and it facilitates the introduction of the eustachian catheter. Descending into the lateral wall of the pharynx from the posteroinferior end of the tubal elevation is the salpingopharyngeal fold (fig. 1), which encloses a muscle of the same name. A second fold, the salpingopalatine, extends anteroinferiorly from the tubal elevation to the so palate. The inferior elevation is called t levator torus or levator cushion [16] and is situat in the floor of the orifice, being directed obliqu anteriorly toward the soft palate. It is due the presence of the levator palati muscle a becomes more prominent when this mus contracts.

THE CARTILAGINOUS PORTION OF THE TUBE

Tubal Cartilages.—The cartilaginous porti of the eustachian tube is composed of muco membrane supported partly by cartilage a partly by a connective tissue layer. This porti of the tube is situated on the basal aspect of th skull, where it lies lodged in a sulcus betwe the greater wing of the sphenoid bone and th petrous portion of the temporal bone (fig. 3 The posterolateral extremity of this portion the tube is firmly fixed to the osseous porti at the level of the medial surface of the spi of the sphenoid bone while its anteromedial end attached to a small tubercle on the posterior ed of the medial pterygoid plate.

The cartilage is in the form of an elongate triangular sheet with its base directed anter medially and with its apex at the isthmus whe it joins the osseous portion (fig. 4). Th cartilage is thickest inferiorly, narrows gradual superiorly and finally becomes a hook of car lage, which bounds the eustachian canal super orly and anteriorly, generally in the shape of shepherd's crook.[13] Two parts are distinguishe in the cartilage of the tube, a medial lamina whi is toward the rear and a lateral lamina which situated superiorly and anteriorly. In a series cross sections the cartilage near the pharyng ostium is right angle shaped (⌐), while th of the tube farther away is crook shaped (⌠ As a result, a groove is produced in the later three fourths of the cartilaginous portion, whi is open inferiorly and laterally. Thus the tub cartilage forms the superior and posterior sup port of the mucous membrane lining, whereas th inferior three fourths of the anterolateral an inferior walls is supported by a dense fibrou membrane called the salpingopharyngeal fascia.

Salpingopharyngeal Fascia.—This dense f brous membrane, the salpingopharyngeal fasci is situated between the tubal muscles and th mucosa (fig. 11). It is attached superiorly to th

15. Gray, H.: Gray's Anatomy, ed. 27, edited by T. B. Johnson and J. Whillis, London, Longmans, Green & Co., 1938.

16. Morris, H.: Morris' Human Anatomy, ed. 1 edited by J. P. Schaeffer, Philadelphia, The Blakiston Company, 1942.

e end of the hooklike lateral lamina of cartilage d, after passing inferiorly and medially between e tensor and levator palati muscles externally d the mucosa internally, is attached to the ferior free end of the medial lamina of carti-

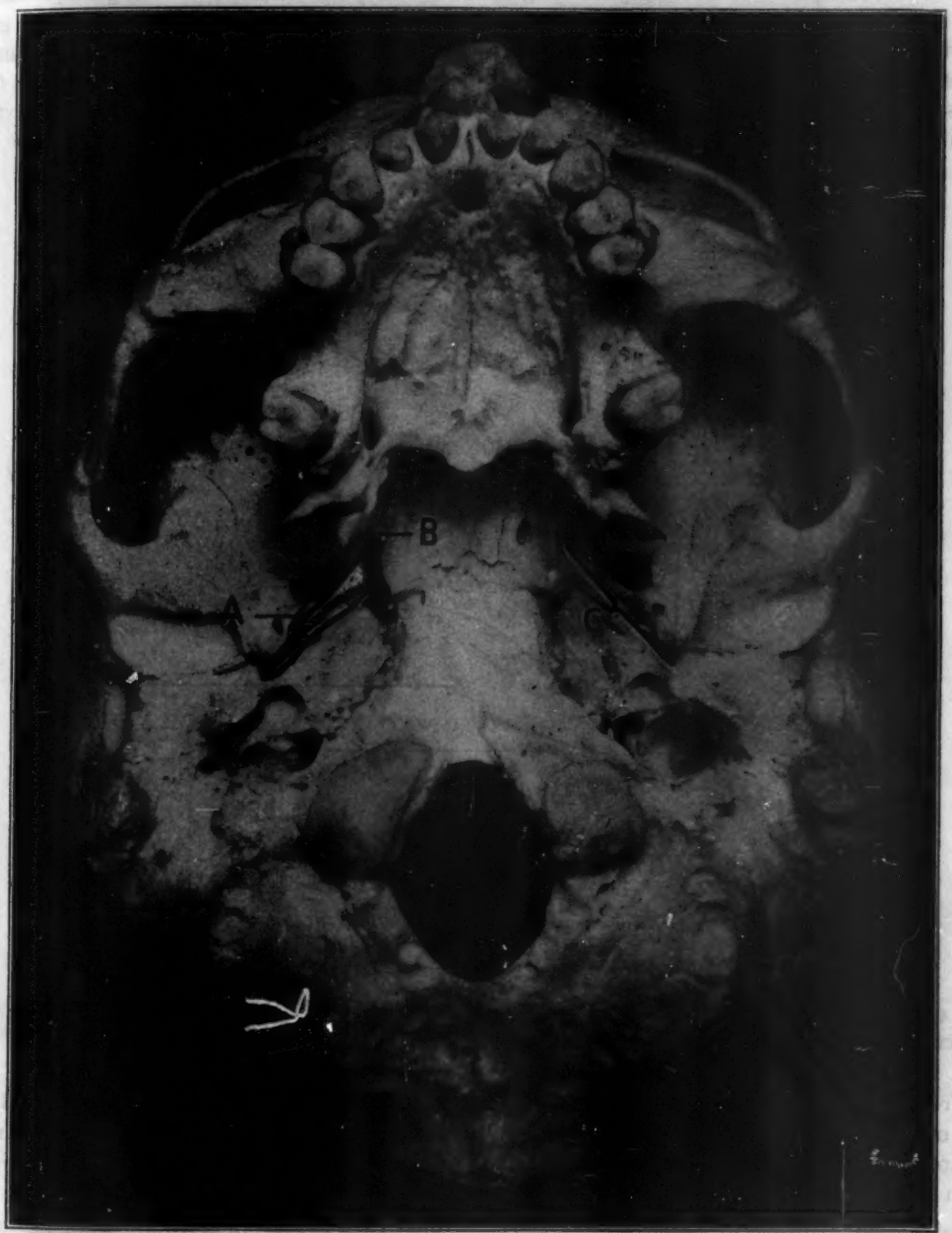


Fig. 3.—A photograph of the basal view of an adult skull with the limits of the sulcus of the eustachian tube ndicated on the right half and the shadow of the tubal cartilage superimposed on the left half. *A* indicates the phenoid spine; *B*, tubercle of the medial pterygoid plate; *C*, the sulcus for the eustachian tube.

ge, blending with the perichondrium which urrounds the accessory cartilages when these are resent, in the floor or the lateral wall of the tube. his fascia, which lends support to this portion f the tube, may represent a thickening of the external portion of the mucosa or, as Citelli [17] suggested, an aponeurotic fascia of the muscles.

Accessory Cartilages. — Numerous authors, notably Henle,[18] Wolff,[19] Urbantschitsch,[18] Citelli [17] and Zuckerkandl,[20] have described accessory cartilages at the anteromedial end of the tubal cartilage. Henle [18] was the first to

17. Citelli, S.: Sulla struttura della tromba d'Eustachio nell-uoma, Arch. ital. di otol. **16**:404-418 and 441-452, 1905.

(Footnotes continued on next page)

observe them. He described several small pieces of cartilage connected to each other by connective tissue or more frequently cartilaginous ridges separated by fissures into which the perichondrium penetrated deeply. Zuckerkandl,[20] who devoted considerable space in his paper to the description of accessory cartilages, claimed that he had observed as many as seven of them in one tube, that they occupy various positions, namely, in the salpingopharyngeal fascia as well as in the superolateral and medial cartilaginous walls of the tube, and that they are

tubal cartilage, some authors have claimed th it is of the hyaline variety while others have sa that it is elastic (fibrocartilage). Urban schitsch[18] expressed the opinion that its stru ture varies with age. Thus in children it hyaline, with the chondrocytes more or le uniformly distributed in the ground substanc whereas in adults the chondrocytes are groupe into island-like clusters, with the ground su stance appearing striated and granular. Citelli agreed that in children the chondrocytes a uniformly distributed but claimed that the groun

Fig. 4.—A drawing of the cartilage of the eustachian tube of an adult in situ (*A*) and isolated (*B*), wit cross-sectional outlines at various levels (*a*, *b*, *c*, *d*).

most frequently observed on the inferolateral border of the tubal cartilage. Our own preparations are illustrated in figure 4, showing their location in the floor of the tube.

Histologic Appearances of the Tubal Cartilage.—As to the microscopic structure of the

substance when properly stained exhibits in mos cases an elaborate network of elastic fibers whic traverse the cartilage in all directions, being mor abundant at the apex than at the base. In th tubal cartilage from a young man he found th elastic network less abundant than in the chil and the chondrocytes congregated into group in a few places, especially toward the base whereas otherwise they were evenly distributed In tubes from 2 elderly men he found th chondrocytes everywhere grouped into islets wit less elastic fibers than in the tube from the youn man, the ground substance between the islet having a granular appearance. Moreover, h

18. Henle, J.: Handbuch der systematischen Anatomie des Menschen, Braunschweig, F. Vieweg u. Sohn, 1866, vol. 2, pp. 753-754.

19. Wolff, D.: Microscopic Anatomy of the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **43**:483-494 (June) 1934.

20. Zuckerkandl, E.: Zur Anatomie und Physiologie der Tuba Eustachiana, Monatschr. f. Ohrenh. **7**:146-150 (Dec.) 1873; **8**:125-132 (Nov.) 1874.

nd calcified cells scattered throughout the rtilage. He claimed, however, that as a rule tubal cartilage has little tendency toward cification. Numerous calcified chondrocytes re present in some of our preparations g. 5).

Histologic preparations of the tubal cartilage m our specimens are illustrated in figure 6. merous fissures can be seen traversing the rtilage at various places, in which are present

the fibers of the tensor palati muscle blend with it, while superiorly (fig. 6*D*) it blends with the periosteum which lines the sulcus on the base of the skull, thus forming a dense fibrous membrane serving to fix the tube in the sulcus (fig. 3). Figure 7 is a high power magnification of an adult's tubal cartilage showing the arrangement of the chondrocytes into groups, which are surrounded by a ground substance presenting a fibrous appearance. This view is

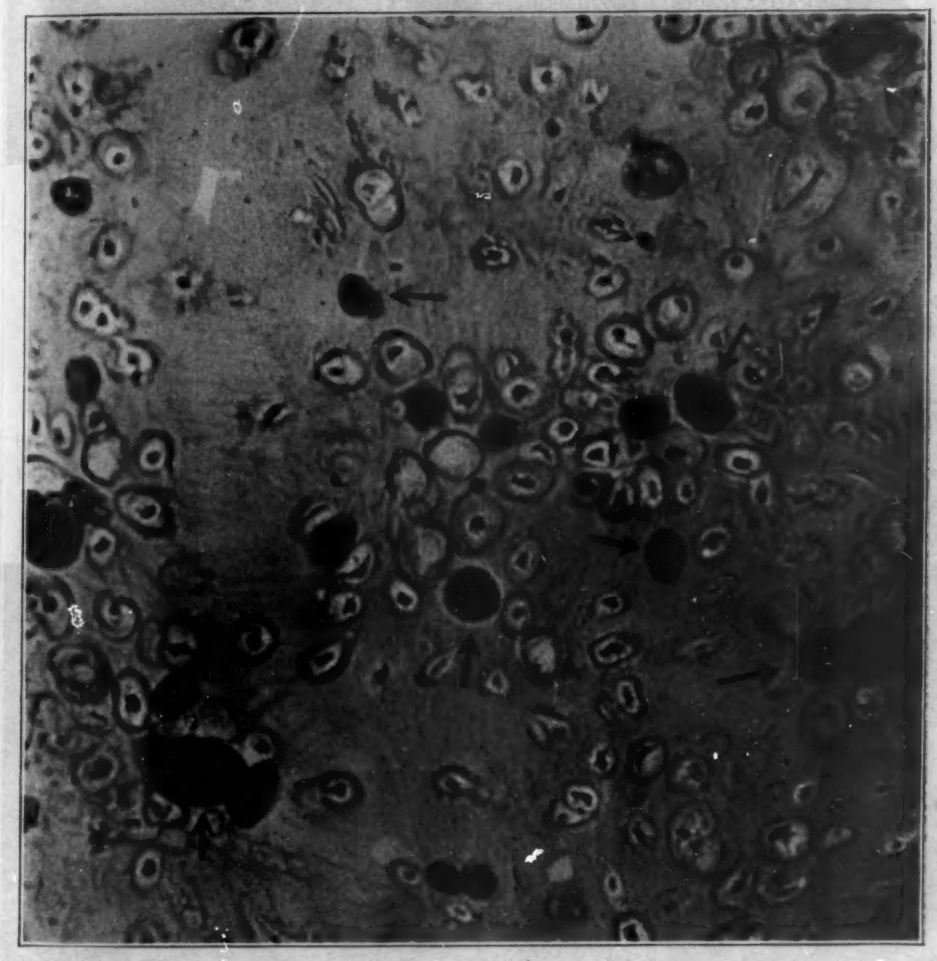


Fig. 5.—A photomicrograph (× 225) of the tubal cartilage of an adult, showing calcified chondrocytes (indicated by arrows).

lands, vessels or loose connective tissue. The rtilage is everywhere surrounded by a well fined perichondrium, which bears the following lations on the various surfaces of the cartilage. hus, on the medial aspect of the medial plate g. 6*A*) the perichondrium lies between the rtilage and the tunica propria of the pharyngeal ucosa; on the lateral aspect of this plate g. 6*B*) and on the medial aspect of the teral plate it lies between the cartilage and e tunica propria of the tubal mucosa; on the teral surface of the lateral plate (fig. 6*C*) typical of the entire cartilage. The chondrocytes of the tubal cartilage from a newborn infant appear more or less uniformly distributed in a matrix which contains abundant fibers. According to Bryant,[8] since the tubal cartilage is elastic, it yields readily to pressure and allows freedom of movement.

Shape of Lumen.—The lumen of the cartilaginous portion of the tube as seen in cross section varies considerably in shape. Near the pharyngeal ostium (fig. 8*A*) it is irregularly triangular with its apex directed superiorly

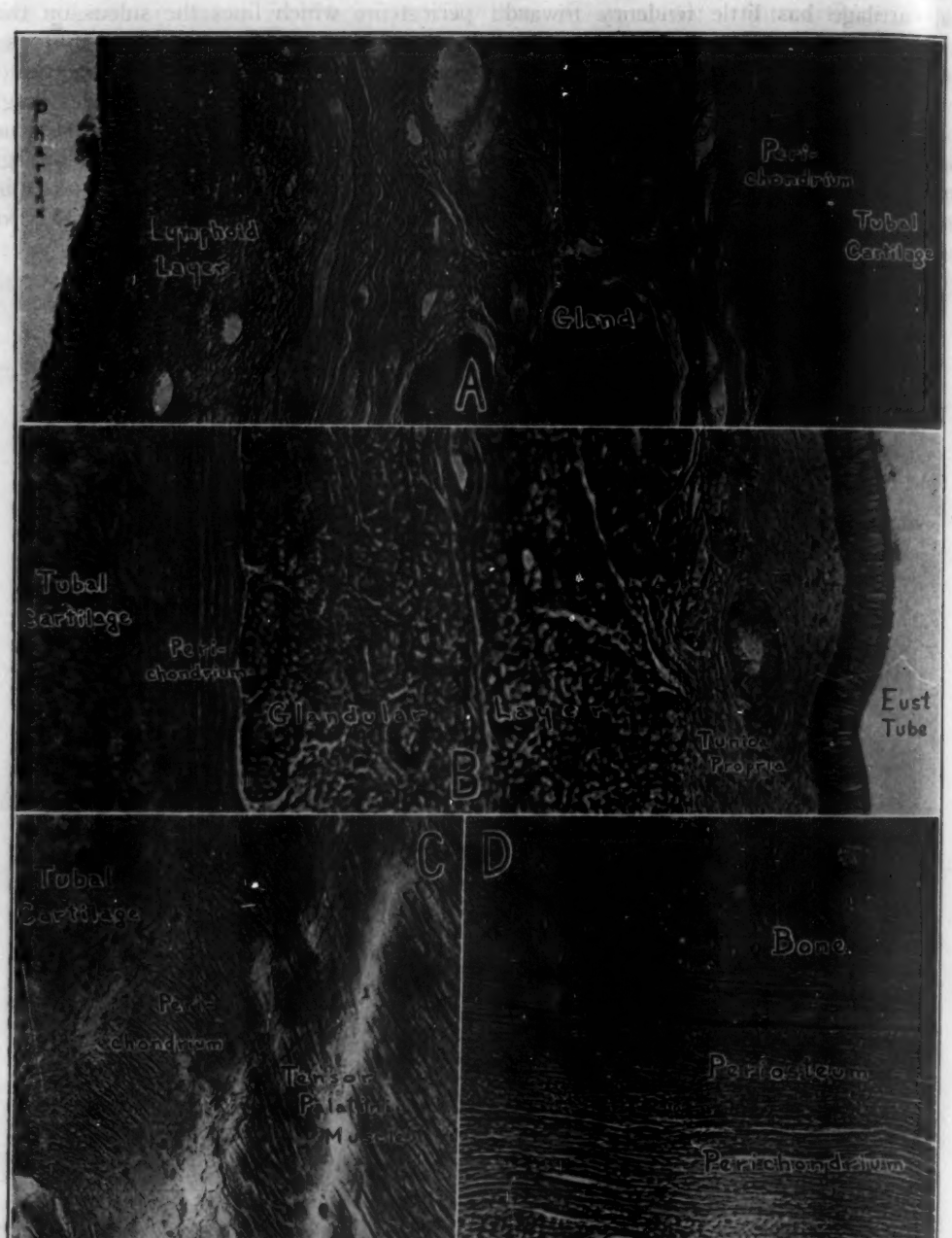


Fig. 6.—Photomicrographs of the perichondrial relations of the tubal cartilage: *A*, posteromedial relati of the medial lamina (× 65). *B*, lateral relations of the medial lamina (× 65). *C*, lateral relations of lateral lamina or hook (× 32.5). *D*, superior relations of the cartilage to the sulcus (× 32.5).

oof); its base, which is marked by the presence folds of mucous membrane, faces inferiorly oor). Traced posterolaterally toward the mus, the lumen assumes an elliptic shape gs. 2 and 8*B*) or one somewhat like an erted comma with its superior one third row and curved laterally and its inferior two rds more rounded and dilated. As the lumen raced farther auralward, it gradually becomes uced to a narrow vertical fissure by the

folds, especially in the floor near the pharyngeal end (fig. 8*A*). According to Polvogt and Babb,[21] the appearance of the mucous membrane varies considerably: It may be smooth throughout; more commonly it presents a corrugated appearance due to the presence of evaginations, which may be simple finger-like projections or more complex longitudinal folds. These authors claimed, moreover, that the folds are more often found in the pharyngeal end, sometimes occurring

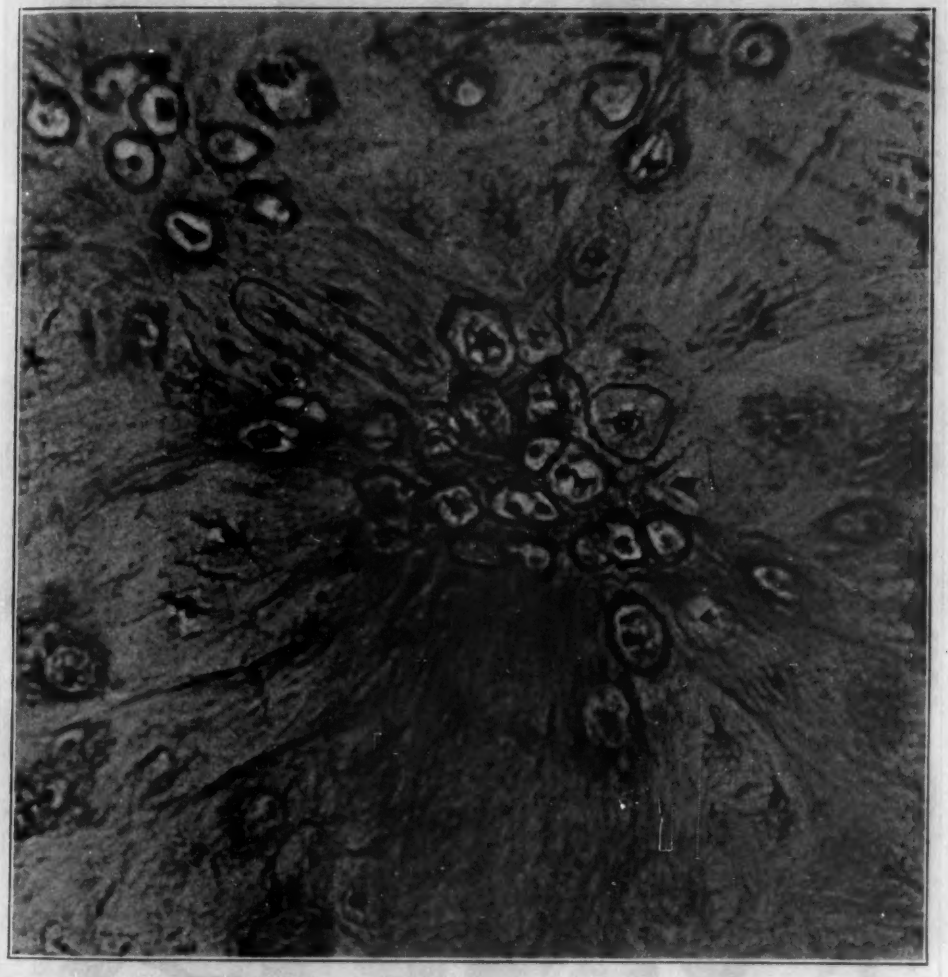


Fig. 7.—A photomicrograph (×235) of the tubal cartilage of an adult, showing the arrangement of ondrocytes in a group surrounded by fibrous-appearing ground substance.

proximation of its side walls (fig. 8*C*), the of and the floor being approximately equal width. Various authors have pointed out that e portion of the lumen which occupies the erval between the lateral and medial cartiginous laminae remains permanently open. At e isthmus (fig. 8*D*) the lumen is still somewhat slitlike, but the vertical height is reduced.

Mucosa.—The mucous membrane in the rtilaginous portion of the tube usually presents irregular appearance due to the presence of

only in this region, but that they may extend throughout the entire length of the tube. Citelli[17] likewise claimed that the mucosa varies not only in different persons and in different parts of the tube but also according to age. Thus he pointed out that near the pharyngeal end the folds are more constant and prominent on the floor and the inferior halves of the walls

21. Polvogt, L., and Babb, D. C.: Histologic Studies of the Eustachian Tube of Individuals with Good Hearing. Tr. Am. Otol. Soc. **31**:41-46, 1940.

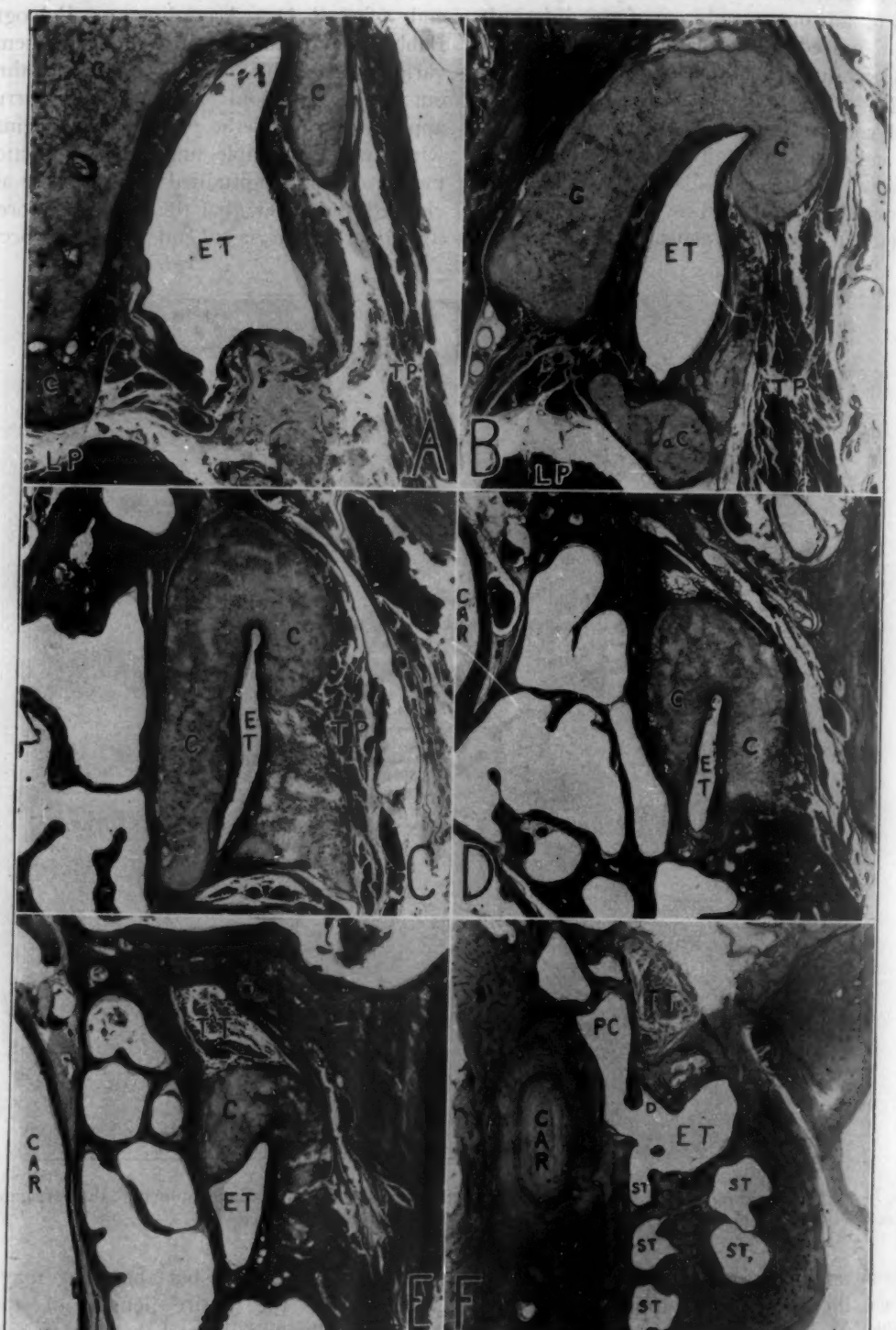


Fig. 8.—Photographs of cross sections of the eustachian tube of an adult: *A*, just lateral to the pharyn orifice; *B*, at the midpart of the cartilaginous portion; *C*, nearing the isthmus from the cartilaginous si *D*, at the isthmus; *E*, on the osseous side of the isthmus; *F*, at the tympanic end of the tube with subti (*ST*) and postcarotid (*PC*) air cells. *ET* is the eustachian tube; *C*, the tubal cartilage, *aC*, the accessory ca lage; *Cr*, the bony crest indenting the tympanic end of the tube; *D*, the dorsomedial ridge formed by the bony cr *Car*, the internal carotid artery; *LP*, the levator palatimuscle; *TP*, the tensor palati muscle; *TT*, the ten tympani muscle.

on the roof; auralward from the pharynx are limited to the floor and lower portions e cartilaginous walls and disappear near the nus, where the mucosa becomes thinner and . This author claimed, moreover, that these s are usually only slightly developed in old

The mucous membrane (fig. 9) consists of epithelium and tunica propria. The epithelium is, for the most part, of the pseudostratified ciliated columnar variety. Polvogt and Babb [21] pointed out, however, that the amount of ciliated epithelium found in different tubes is exceedingly

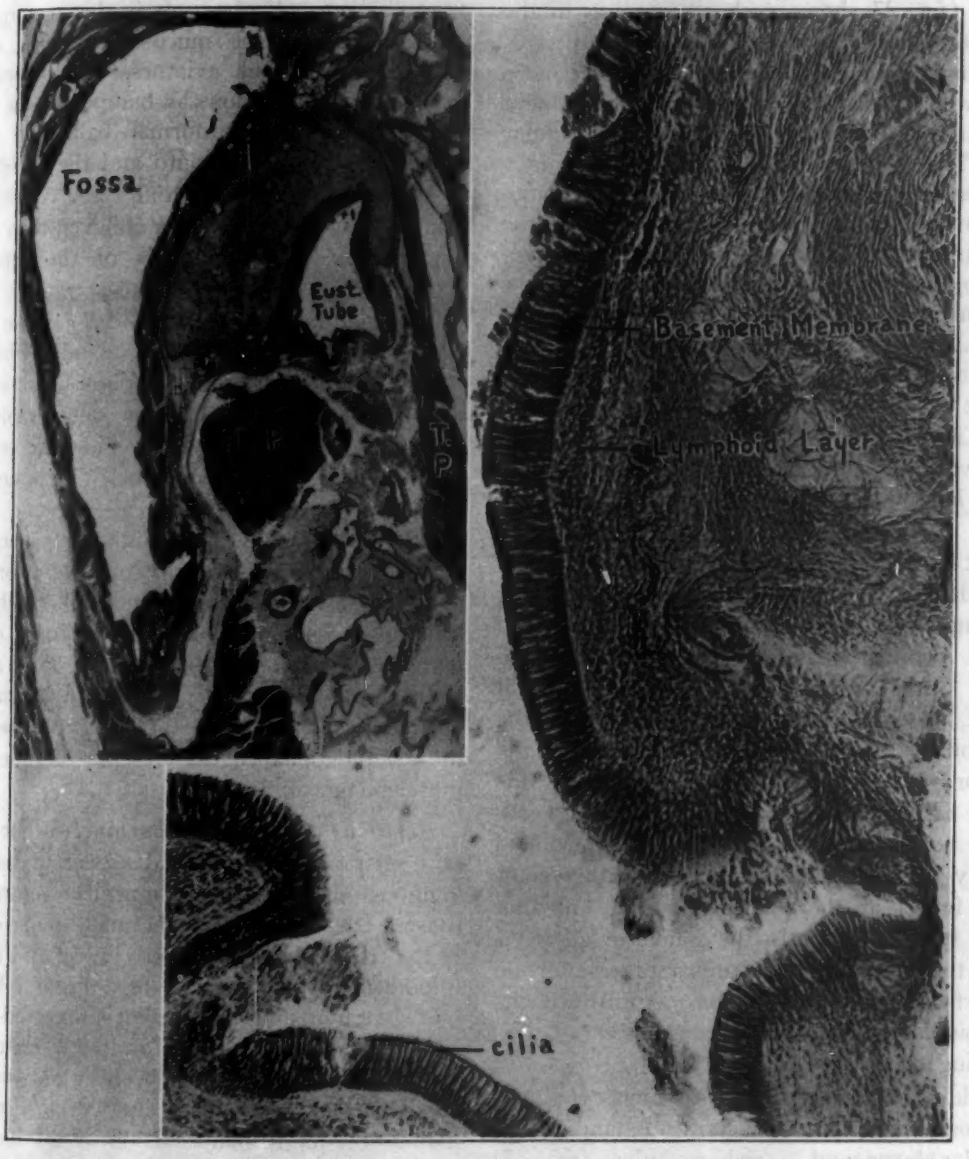


Fig. 9.—A photomicrograph of the mucous membrane of the floor (see inset) of the pharyngeal end of the stachian tube.

sons and in the newborn, whereas in a month old baby he observed a tube with the e end of a longitudinal fold reaching to well r half the height of the lumen. His explana- n of the presence of the folds is that they are med not simply by the folding of the mucosa rather by the development of the adenoid ue contained within them.

variable, that it is always found in the pharyngeal end and may even extend into the middle ear. According to these authors, it is more often lacking on the lateral wall of the tube than on the medial wall. In our preparations cilia are apparently lacking on the roof and superior one half of the medial wall, where the height of the cells is greatly reduced. Goblet cells are interspersed

between the epithelial cells and appear more numerous in the floor and the lower one half of the medial and lateral walls than on the vault. Polvogt and Babb [21] claimed that whereas large numbers of these cells are present in some tubes, "in others there are only a few." At various places throughout the epithelium can be seen openings of ducts of the glands lying in the tunica propria (fig. 27 shows such an opening in the eustachian tube of a newborn infant).

The tunica propria is divisible into three more or less well defined layers, namely, the basement membrane, a lymphoid layer and a glandular layer. The basement membrane (fig. 9) is a thin stratum of reticular connective tissue immediately underlying the epithelium throughout the cartilaginous portion of the tube. It is penetrated by the ducts of the glands located in the glandular layer as they pass to the surface epithelium. The lymphoid layer (fig. 9) varies in different portions of the tube and, according to most observers, with the age of the subject. Thus Citelli [17] claimed that this layer is not as well developed in the last months of intrauterine life as in childhood and that in old age it is greatly reduced, being almost entirely nonexistent in the superior half of the walls. It is better developed at the pharyngeal end, where the lymphocytes are sometimes collected into follicles forming what is frequently referred to as the "tubal tonsil" (of Gerlach). Citelli claimed, as previously pointed out, that the collection of this adenoid tissue produces the rugae or folds of the mucosa encountered in this portion of the tube. Polvogt and Babb,[21] observing normal ears of all age groups, concluded not only that a lymphoid nodule is rather commonly found at the pharyngeal end but that similar nodules may also be found in all parts of the tube either in the tunica propria or on the surface of the epithelium. Auralward the lymphoid layer gradually becomes thinner, disappearing completely at the vault.

In addition to the presence of lymphocytes, this layer contains numerous ducts from glands in the glandular layer and a rich vascular (figs. 24 and 25) and lymphatic network.

Bezold [22] has taken advantage of this vascular plexus by causing its engorgement with insufflation of salicylic acid in cases of a continuously open tube. The salicylic acid, acting as an irritant, causes an inflammatory swelling of the mucosa, which closes the tube. The patient within a few minutes experiences complete re from his symptoms of autophonia and hyp acusis, "blowing or loud roaring in the ea (synchronous with breathing) and low-pitc tinnitus. This relief usually lasts one or weeks. Colds cause similar relief in patients w continuously open tubes but troublesome stu ness and impairment of hearing for low tones otherwise normal tubes. It is this engorgem and swelling of the mucosa which is rig guarded against in aviators, for it leads dangerous complications by hampering the ten palati muscle in its normal balancing of pressures within the mouth and the middle e

According to Polvogt and Babb,[21] "this v cularity is usually greater in children than adu but it varies in individuals of the same group."

The glandular layer (fig. 6*B*) is so ca because of the abundance of glands, which of the compound tubuloalveolar (mucosero variety. These glands vary considerably w age and with persons as well as in different pa of the walls of the same tube. They are m abundant near the pharyngeal end and on medial wall, where they continue to the va Auralward they disappear from the floor, va and lateral wall. In the vault where they lacking the basement membrane fuses direc with the perichondrium, and in the floor and lateral wall they are replaced with areolar c nective tissue which is intimately related w the salpingopharyngeal fascia of the tu muscles (fig. 6).

Relations of Clinical Importance.—The imp tant relations of the lateral aspect of the ca laginous portion of the tube are the tensor pa muscle (figs. 1 and 10), the mandibular divisi of the trigeminal nerve (figs. 11, 12 and 13), middle meningeal artery (figs. 12 and 13), otic ganglion and the medial pterygoid mus (figs. 1 and [inset], 10, 11 and 12). The me pterygoid relationship has been linked with cli cal situations [23] in which an overclosure of mandible (due to malocclusion or toothlessnes has resulted in compression of the cartilagino portion of the eustachian tube by the short fatter belly of the muscle. Posteromedially tube lies in relation with the petrous part of temporal bone, the levator palati muscle and pharyngeal recess of Rosenmüller (fig. 10). T retrotubal mucosa (fig. 6*A*), which interve

22. Bezold, cited by Shambaugh, G. E.: Continuously Open Eustachian Tube, Arch. Otolaryng. **27**:420-425 (April) 1938.

23. Willhelmy, G. E.: Ear Symptoms Incidental Sudden Altitude Changes, and the Factor of Ov closure of the Mandible: Preliminary Report, U. Nav. M. Bull. **34**:533-541 (Oct.) 1936.

een the recess and the medial tubal lamina, ents the same appearance as the tubal mucosa the exception that lymphoid follicles are abundant and pronounced.

from its junction with the cartilaginous portion to the tympanic ostium. The osseous portion lies on a more horizontal plane than the cartilaginous, the two forming an obtuse angle of

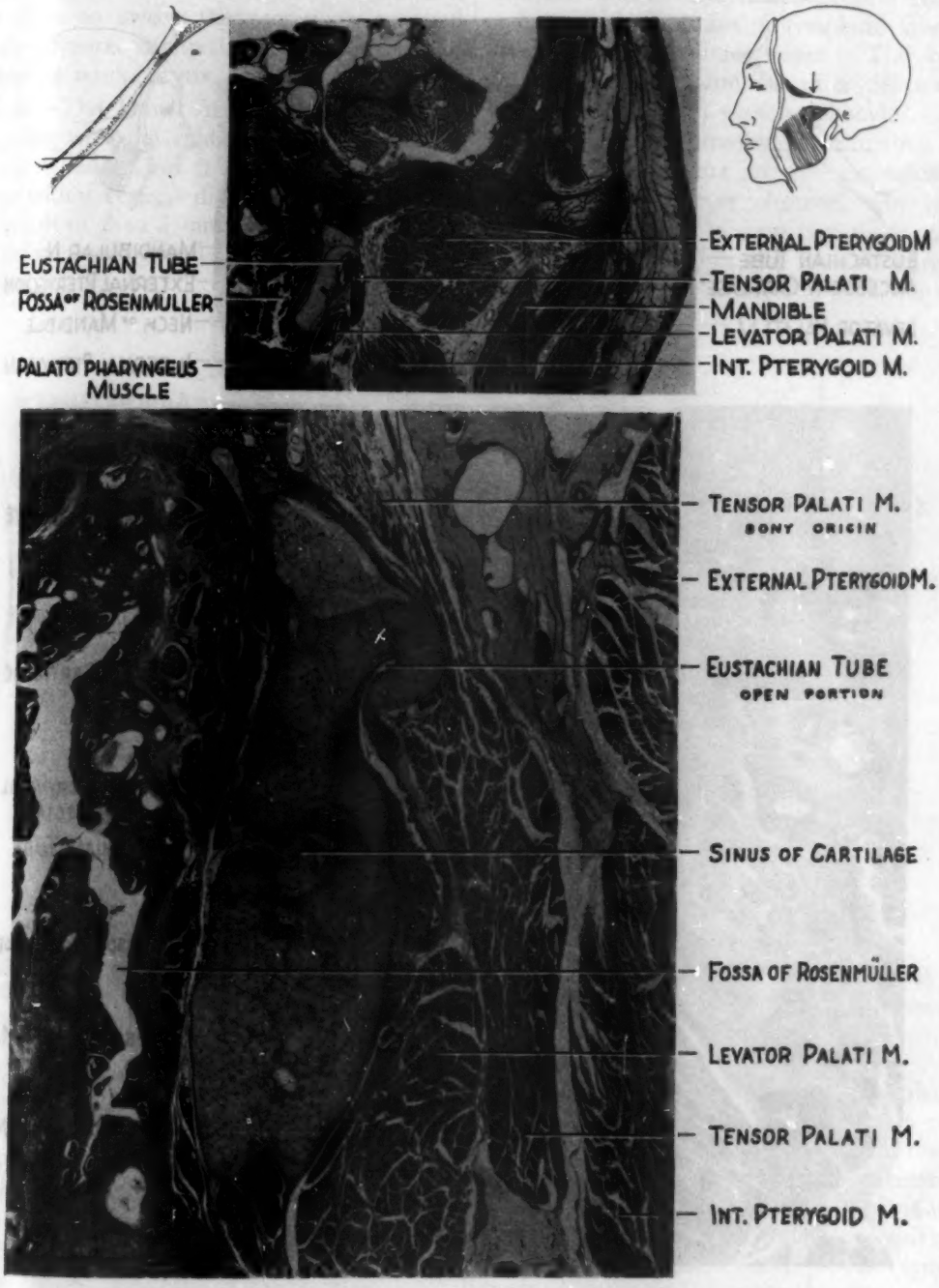


Fig. 10.—Photographs of frontal celloidin sections (40 microns) of the pharyngeal end of the cartilaginous tion of the eustachian tube showing the relations in the adult.

THE OSSEOUS PORTION OF THE TUBE

This division is situated within the petrous rtion of the temporal bone, where it occupies emicanal (semicanalis tubae auditivae) which directed laterally, superiorly and posteriorly about 160 degrees opening outward at their junction. This junction (isthmus) is in the region of the sphenopetrosal fissure at the level of the medial surface of the angular spine of the sphenoid bone (fig. 3).

Isthmus.—The demarcation point of the isthmus is variously interpreted by different authors. In this paper it is taken as that point where bone is formed inferior to the tube (fig. 8 *D*), since in most cases cartilage is found in the superior portion of the tube and may extend as far as the tympanic cavity (figs. 8 *E* and 13). In other cases it may be present on the medial and the lateral as well as on the superior side (fig. 8 *D*). According to Wolff,[19] Henle and Rüdinger were the first to point out that the anteromedial of the osseous portion is jagged or dentated opens obliquely anterolaterally at its junc with the tubal cartilage. As a result the isth is bounded posteromedially and inferiorly bone and anterolaterally and superiorly by c lage. The tympanic ostium is located on anterior wall of the tympanic cavity about 4 (⅙ inch) above the lowest part of the floo the cavity, a point which corresponds to

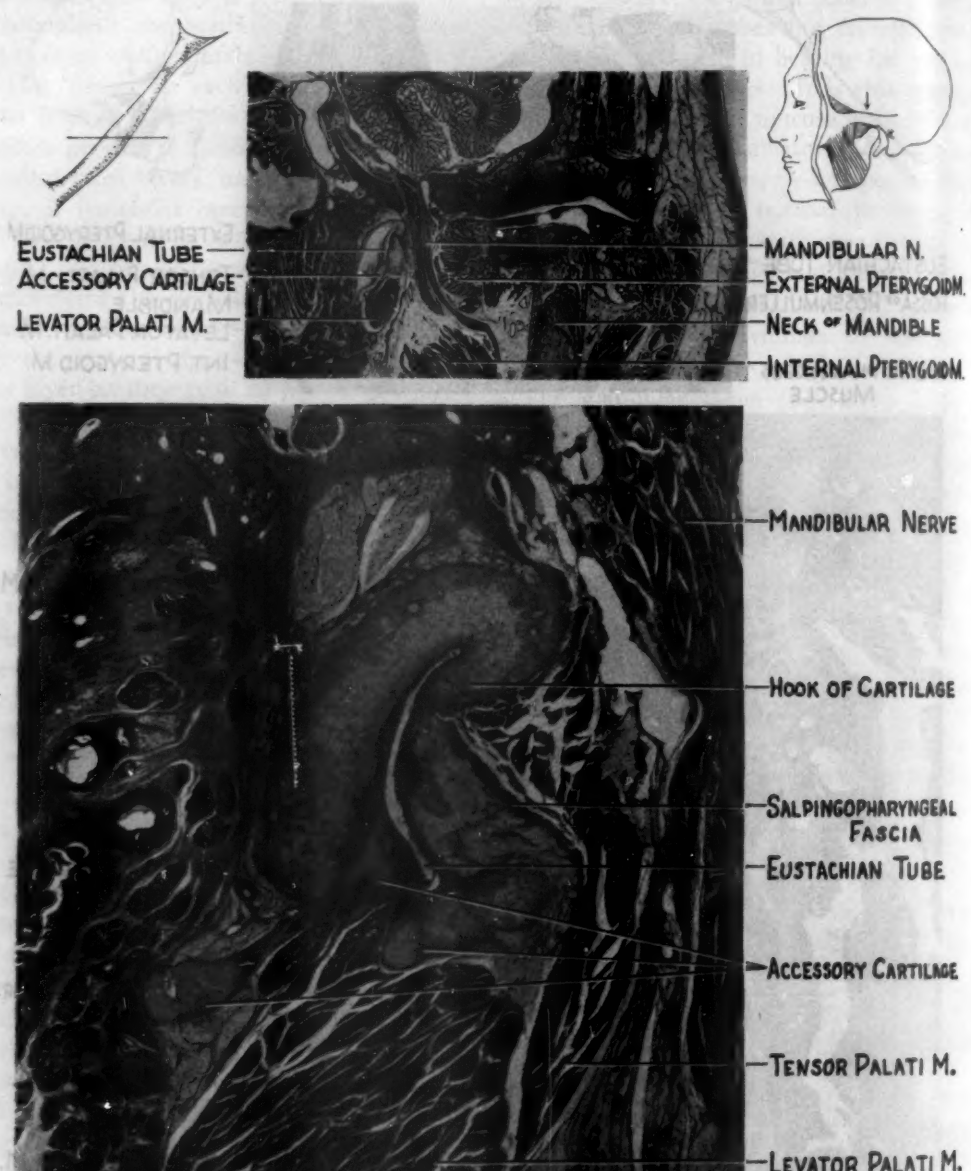


Fig. 11.—Photographs of frontal celloidin sections (40 microns) of the middle of the cartilaginous po of the eustachian tube of an adult, showing the relation of the accessory cartilage, the mandibular nerve, levator and tensor palati muscles and the temporomandibular joint.

erosuperior quadrant of the tympanic membrane. This location is obviously not advantageous for gravity drainage from the cavity in the upright position, although this fact is minimized by the ciliated mucous membrane, which tends to sweep the secretions outward from the tympanic cavity through the eustachian tube into the nasopharynx.

Lumen.—The lumen of the osseous portion corresponds with the caliber of the bony canal in which it lies. Thus it is roughly triangular in shape with a vertical diameter of 3 to 4 mm. and a width of 2 to 3 mm. (fig. 2). The tympanic ostium leading into this triangular-shaped canal is a funnel-shaped opening, 3 to 5 mm.[12] in diameter, in the upper third of the tympanic cavity. The lumen gradually narrows to the isthmus, where it is 2 to 3 mm. in height and 1 to 1.5 mm. in width. Near the tympanic orifice a crest appears in the superior wall toward the medial side, giving the appearance of a diverticulum pointing superiorly (fig. 2).

Mucosa.—The mucous membrane of the osseous portion of the tube (fig. 8 *F*) is thinner than that of the cartilaginous portion and fuses intimately with the periosteum, forming a mucoperiosteal lining. Since the mucous membrane is supported everywhere by bone and since it firmly adheres to the periosteum, the lumen is normally patent. The mucous membrane may be thrown into folds, which, however, are not as numerous and are more delicate than those in the cartilaginous portion. The epithelium is of the low columnar ciliated type, the cells in the vault being somewhat lower than those of the floor and the walls. The tunica propria consists of a basement membrane, a lymphoid layer and a loose connective tissue layer. The lymphoid layer is thin and consists of a delicate elastic reticulum infiltrated with lymphocytes. Citelli,[17] who made an extensive investigation of the microscopic structure of the eustachian tube, claimed he had never observed true lymphoid follicles here except in some pathologic specimens and that in old people this layer is barely indicated. The loose connective tissue layer supplants the glandular layer of the cartilaginous portion, since glands are scarce in this region. A rich vascular plexus can be seen in the floor and walls in this layer as well as a rather sizable nerve (fig. 8 *D-F*) in the floor. Both Citelli[17] and Wolff[24] mentioned and illustrated this nerve. It is a branch of the tympanic plexus (tympanic branch of the glossopharyngeal nerve).

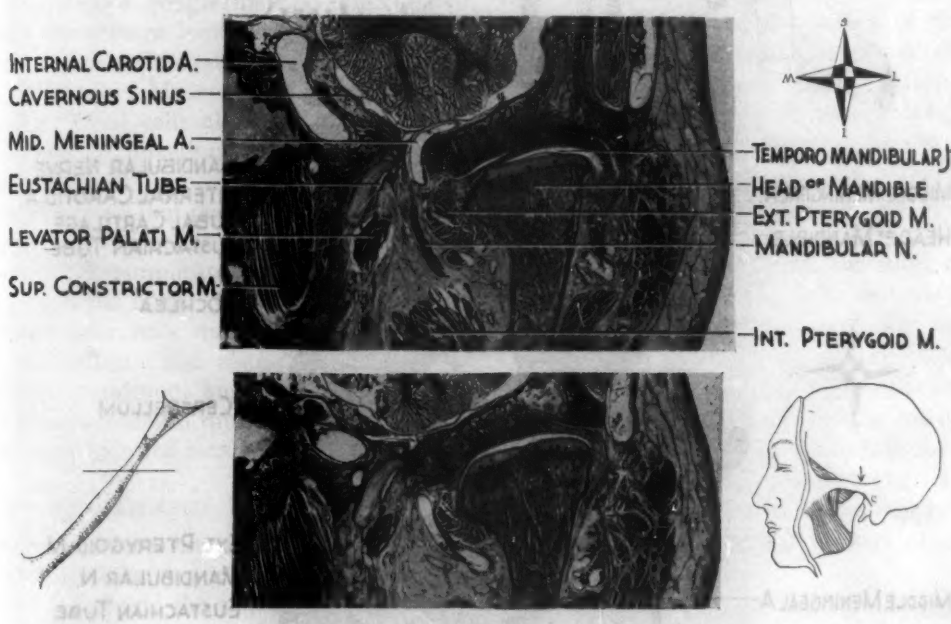


Fig. 12.—Photographs of frontal celloidin sections (40 microns) near the isthmus of the eustachian tube in adult, showing the relation to the middle meningeal artery, the mandibular nerve and the external and internal pterygoid muscles.

Relations.—The relations of the osseous portion become significant through the potential connections of vascular and lymphatic systems of juxtapositioned structures. Superiorly in the posterior two thirds of this portion lies the semi-

24. Wolff, D.: Microscopic Observations of the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **40**:1055-1067 (Dec.) 1931.

canal for the tensor tympani muscle (figs. 1 and 8 *E-F*), which is separated from the osseous portion of the eustachian tube by means of a thin septum of bone. Posteromedially lies the carotid canal (figs. 8 *D-F* and 13). According to Quain [11] and others, the carotid canal is occasionally found to communicate with this portion of the tube.[11]. Bast and Forester [25] stated that subtu air cells were present in 88 per cent of fetuses over 27 weeks old and in 90 per c of 27 infants up to 6 years and that they ope most often into the middle ear cavity, altho occasionally they opened into the tube (8 *F*). Polvogt and Babb [21] hold that in mar

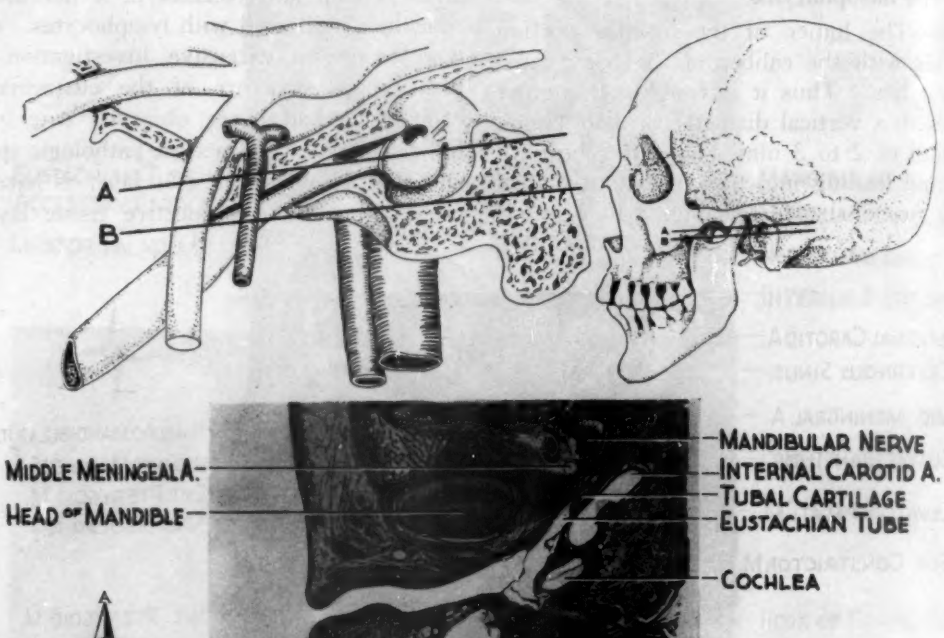


Fig. 13.—Photographs of horizontal celloidin sections (40 microns) illustrating the relations of the eu chian tube of the adult to the internal carotid artery, the temporomandibular joint, the middle and the i ear, the mandibular nerve and the middle meningeal artery.

of the tube, "owing to deficiencies in the bony septum between them." Anterolaterally lies the tympanic part (the posteromedial boundary of the mandibular fossa) of the temporal bone (figs. 1, 12 and 13). Inferiorly near the tympanic end lie subtubal air cells, which may either communicate with the middle ear cavity or with the pneumatization of the pars petrosa the t cells are continuous with the tube. Lindsa

25. Bast, T. H., and Forester, H. B.: Origin Distribution of the Air Cells in the Temporal B Arch. Otolaryng. **30**:183-205 (Aug.) 1939.

26. Lindsay, J. R.: Petrous Pyramid of Tem Bone, Arch. Otolaryng. **31**:231-255 (Feb.) 1940.

d in the 21 per cent of heads which showed matization of the petrous apex that two s of them had tubal cells coming from the rior part of the tympanum and the eustachian and he gave these facts surgical import by ng that "in these cases [of anterior petrositis] only feasible and rational approach is by way e middle ear and the mouth of the eustachian ." His other pertinent clinical observations that (1) simple peritubal cells which do not nd into the apex tend to drain freely and therefore seldom the cause of complications cute suppuration and (2) that none of these tubal cells are demonstrable by roentgenons.

olvogt and Babb [21] mentioned that dilation he tube with a bougie may be difficult and rdous when there is marked pneumatization many tubal cells continuous with the main ion of the tube. Moreover according to these hors the "tubal cells allow an easy pathway the spread of infection throughout the temal bone."

Wolff [24] claimed that peritubal cells may be as e as or larger than the main lumen. They do normally communicate with the main tube are diverticula of the tympanum. The lumens he pneumatic cells may become lined with ted epithelium. She also pointed out that athologic conditions suppuration may break ugh from a diseased tube into these cells and then pass forward into the petrous apex.

MUSCLES RELATED TO THE TUBE

Three muscles are intimately associated with eustachian tube, namely, the tensor palati, levator palati and the salpingopharyngeus.

Tensor Palati Muscle.—Situated on the anteroral aspect of the membranocartilaginous por of the tube, the tensor palati (tensor veli atini of Politzer; sphenosalpingostaphylinus; umflexus palati of Politzer; abductor tubae Tortual; dilator tubae of von Tröltsch and dinger) takes origin (fig. 1) from the spine of sphenoid bone, the scaphoid fossa, the lateral ina of the tubal cartilage, the posterior half of membranous wall of the tube [27] and the ingopharyngeal fascia. Its flat belly runs nward and forward, lateral to the pharyngoilar fascia, outside the upper margin of the erior constrictor muscle, obliquely across the ral wall of the membranous tube and medial ygoid plate, to the hamulus of this same plate, und which its tendon turns medially toward its radial insertion into the soft palate. Those fibers which arise from the tubal cartilage and the salpingopharyngeal fascia were termed by Rüdinger the dilator tubae muscle.[28] According to Wolff,[19] Rüdinger, von Tröltsch and Mayer described a direct passage of the tensor palati into the tensor tympani muscle. No evidence of this relationship was found in our microscopic preparations. The tensor palati muscle is supplied by the mandibular division of the trigeminal nerve (fig. 14) by way of a branch from the otic ganglion.[29]

Levator Palati Muscle.—This muscle, situated posterolateral to the eustachian tube and then inferior to its pharyngeal orifice, takes origin from a quadrate area on the inferior surface of the apex of the petrous portion of the temporal bone just anterior to the orifice of the carotid canal and from the lower border of the medial lamina of the tubal cartilage (rarely, if ever, from the tube itself). It passes obliquely, inferiorly and medially anterior to the salpingopharyngeal muscle (fig. 15) and parallel with the tubal cartilage, thence below the pharyngeal orifice of the tube, where it produces (particularly when contracted) an elevation called the levator torus (fig. 1). The levator palati is separated from the tensor palati (in order from posterior to anterior) by the eustachian tube, (figs. 1 and 15), the pharyngobasilar fascia (fig. 15), the superior fibers of the superior constrictor muscle and the anteroinferior fibers of the palatopharyngeal muscle (fig. 15). The levator palati receives its nerve supply from the long looplike pharyngeal branch of the vagus nerve (fig. 14).

Salpingopharyngeus Muscle.—Situated posterior to the pharyngeal orifice of the tube (fig. 15), the salpingopharyngeus muscle takes origin by small slips of muscular and tendinous fibers from the medial and inferior corner of the tubal cartilage.[27] Occasionally fibers arise from the medial one-half inch (1.2 cm.) of the inferior border [27]; rarely they are found to arise from the entire inferior border of the cartilage [10] or from an accessory nodule of cartilage in the region.[27] The muscle extends downward and slightly backward for 1½ inches (3.8 cm.) as a separate bundle and then blends with the anterior and middle fibers of the palatopharyngeus, which in turn insert into the superior cornu of the thyroid cartilage and the lateral and posterior walls of the pharynx, respectively. However,

7. McMyn, J. K.: The Anatomy of the Salpingoryngeus Muscle, J. Laryng. & Otol. **55**:1-22 (Jan.)

28. Quain.[11] von Tröltsch.[12]

29. Rich, A. R.: The Innervation of the Tensor Veli Palatini and Levator Veli Palatini Muscles, Bull. Johns Hopkins Hosp. **31**:305-310 (Sept.) 1920.

no fibers of the salpingopharyngeus itself reach the superior cornu.[27] The salpingopharyngeus likewise receives its nerve supply from the pharyngeal branch of the vagus nerve (fig. 14).

Action of These Muscles.—As to the general action of these muscles related to the eustachian tube, the tensor and levator palati muscles, as their names imply, act on the soft palate, the former making it tense and the latter elevating it. Since the salpingopharyngeus muscle is considered as a fasciculus of the palatopharyngeus muscle, it is said to approximate the posterior tonsillar pillars,[30] to depress the soft palate,[16] to elevate the pharynx [31]—and very questionably to elevate the larynx [16] and to draw the medial plate of the tubal cartilage slightly posteriorly, thereby retracting the tube, although it is admitted that the muscle is weak and may be lacking.[10] Rich,[3] however, from the point of view of experiments on dogs, whose anatomy in this region is very similar to that of man in contradistinction to the anatomy of sheep [30] and that of birds,[32] stated that stimulation of the palatopharyngeus causes no change in the tubal orifice and that

> no muscle, except the tensor palati, exerts any influence upon the pharyngeal orifice of the Eustachian tube, and there is no suggestion that the other muscles of the region act as auxiliaries or antagonists to the tensor in its function of opening the tube.

Prior to the time of Rich, the action of t muscles on the eustachian tube was determi either by pure speculation from anatomic or and insertions or by cadaveric experimentati Rich [3] concluded that

> the tensor palati is the only muscle which is functio related to the Eustachian tube. Contraction of muscle is always accompanied by a dilatation of tubal orifice and lumen. . . . Relaxation of the t palati is followed by a passive return of the tubal to the condition of approximation which they norm occupy when at rest.

It may be pointed out that the apposition w at rest is never complete, because of the conca at the point of junction of the medial lateral laminae of the cartilage.[29] Rich s stantiated his experimental data on dogs by m

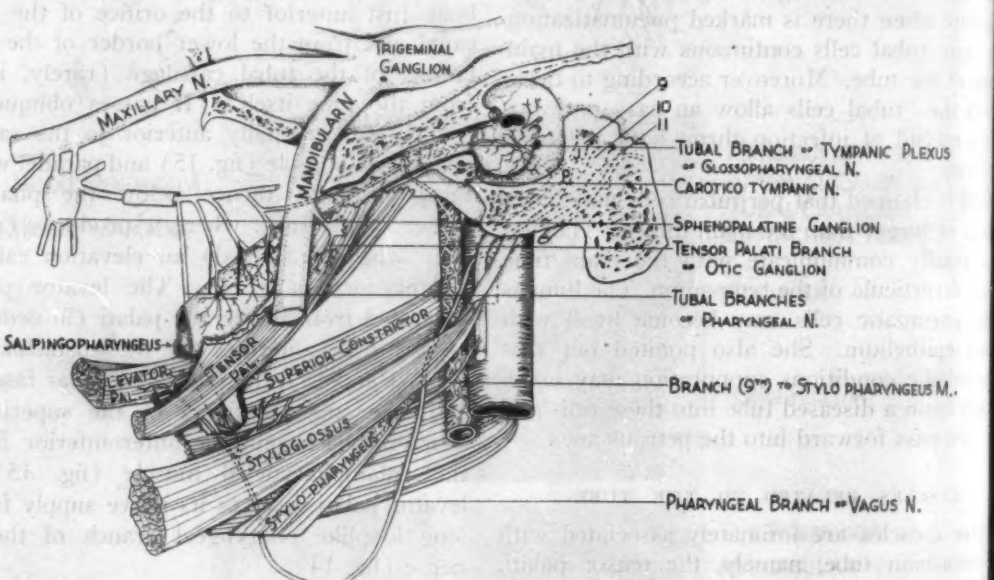


Fig. 14.—A drawing of a lateral view of the nerve supply of the eustachian tube and its related muscles.

pharyngoscopic observations on a man suffer from unilateral paralysis of the tensor pa muscle following disease of the fifth nerve. observed that "the pharyngeal orifice of the t on the affected side appeared somewhat m patulous than that on the normal side" and t during deglutition "the orifice on the side the tensor paralysis remained quite stationa although the levator palati, palatopharyng and superior constrictor muscles were contra ing vigorously." Rich likewise narrowed many voluntary acts suggested to open the or and lumen of the tube to three—swallowi yawning and sneezing. Advantage of these th voluntary acts is taken by aviators during fli to equalize the pressure on the tympanic me brane during rapid changes of altitude.

30. Yule, C. T. F.: On the Mechanism of Opening and Closing the Eustachian Tube, J. Anat. & Physiol. **8**:127-132 (Nov.) 1873.

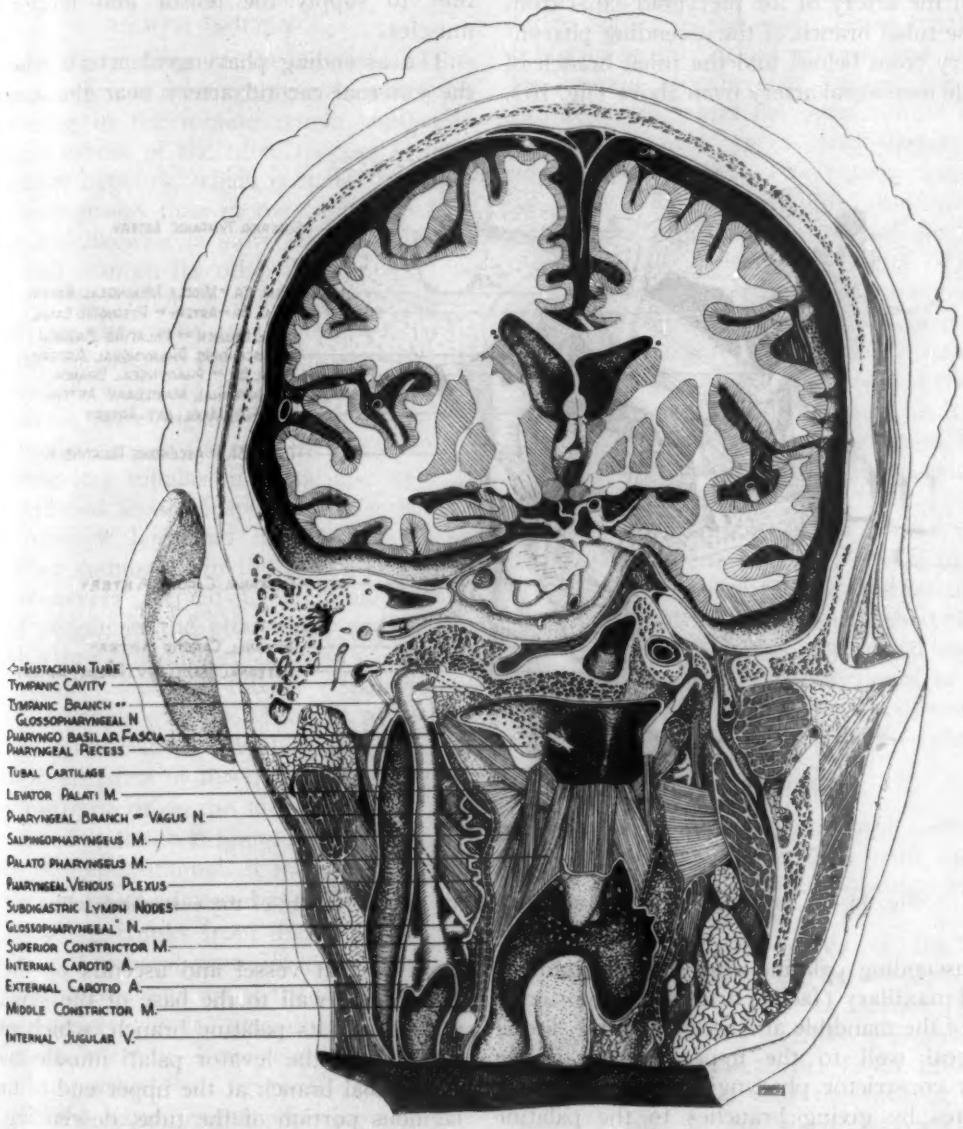
31. Gray.[15] Morris.[16]

32. Toynbee, J.: On the Muscles Which Open the Eustachian Tube, Proc. Roy Soc., London **6**:286-287 (Feb. 17) 1853.

33. Bryant.[8] Huschke.[10]

me of the earlier observers contributed rate data on the applied physiology of the achian tubes, although their anatomic ex- ations have proved fallacious. Toynbee[32] the first to state the proposition that the achian tube is normally closed and that it is ed only during the act of swallowing. His words are as follows:

> that the feeling of pressure in the ears does not disappear, but it remains until the act of deglutition is again performed while the nose is closed. . . . That the act of deglutition opens the Eustachian tubes, was inferred also from the custom usually adopted of swallowing while the descent in a diving bell is performed; by this act the condensed air is allowed to enter the tympanum, and the sensation of pain and pressure in the ears is removed or entirely avoided.



ig. 15.—A drawing of the posterior surface of a section of an adult head made through the fossa of mmüller, with the muscles of the soft palate and oral pharynx dissected.

, that the act of swallowing is the means whereby Eustachian tubes are opened, is shown by some riments of which the following may be cited. If mouth and nose be closed during the act of swallow- the saliva, a sensation of fullness or distention is uced in the ears; this sensation arises from the which is slightly compressed in the fauces, passing and distending the tympanic cavities: upon re- ng the hand from the nose, it will be observed

Yule[30] twenty years later called attention to the sound which accompanies the opening of the eustachian tube:

> It is a sharp, crackling sound, which is referred to some part of the tympanum, or perhaps to the membrane itself. This, I have no doubt whatever, is caused by the separation of the walls of the Eustachian tube; . . . The sound can be heard by anybody at the

commencement of the act of swallowing, and forms the prelude to a series of noises arising from the moving of mucus, etc., about the posterior nares and pharynx.

BLOOD SUPPLY

The eustachian tube is supplied by the following arteries (from anterior to posterior): the ascending palatine artery from below, the tubal branch of the pharyngeal branch of the internal maxillary artery from above, the tubal branch of the artery of the pterygoid canal from above, the tubal branch of the ascending pharyngeal artery from below, and the tubal branch of the middle meningeal artery from above (fig. 16).

The artery of the pterygoid canal arises f the internal maxillary artery within the pter palatine fossa and passes backward through pterygoid canal to the nasopharynx, whe gives off its tubal branch, which descends t superior surface of the tube, thereupon divi into two branches, one supplying the m medial surface of the cartilaginous portio the tube and the other descending lateral t tube to supply the tensor and levator p muscles.

The ascending pharyngeal artery arises f the external carotid artery near the comm

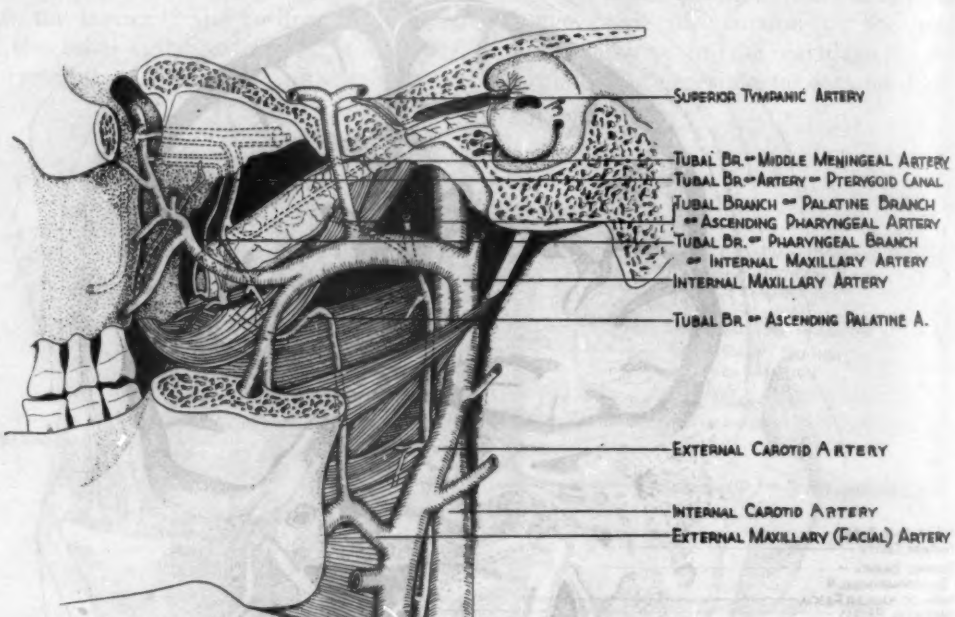

Superior Tympanic Artery
Tubal Br. of Middle Meningeal Artery
Tubal Br. of Artery of Pterygoid Canal
Tubal Branch of Palatine Branch of Ascending Pharyngeal Artery
Tubal Br. of Pharyngeal Branch of Internal Maxillary Artery
Internal Maxillary Artery
Tubal Br. of Ascending Palatine A.
External Carotid Artery
Internal Carotid Artery
External Maxillary (Facial) Artery

Fig. 16.—A drawing of the lateral view of the arterial supply of the eustachian tube.

The ascending palatine artery arises from the external maxillary (facial) artery at the posterior border of the mandible and ascends on the lateral pharyngeal wall to the upper border of the superior constrictor pharyngis muscle, where it terminates by giving branches to the palatine tonsil, the soft palate and the eustachian tube. The tubal branch divides to send smaller vessels to both sides of the tube near the pharyngeal orifice.

The pharyngeal branch of the internal maxillary artery arises within the pterygopalatine fossa and runs backward through the pharyngeal canal to the roof of the nasopharynx, where it gives off the tubal branch, which descends and is distributed to the medial wall of tube near the pharyngeal orifice.

ment of that vessel and ascends on the la pharyngeal wall to the base of the skull, w it gives off its palatine branch, which runs ward along the levator palati muscle and g off a tubal branch at the upper end of the c laginous portion of the tube, descending on inferolateral surface.

The middle meningeal artery arises from internal maxillary artery (fig. 16) and pa superiorly on the tensor palati muscle cove the lateral end of the cartilaginous portio the tube. Just before it enters the skull thro the foramen spinosum it gives off the t branch, which divides and supplies both the tilaginous and the osseous portion of the t As the osseous division of this tubal bra emerges from the osseous semicanal of the te

pani muscle it anastomoses at the tympanic ce of the tube with the superior tympanic ry. Both of these arteries are branches of middle meningeal artery, which gives off the l branch just before it enters the skull and superior tympanic just after it passes through foramen spinosum.

he venous drainage is maintained through pterygoid plexus of veins.

LYMPH SUPPLY

eneath the mucosa of the eustachian tube a most efficient drainage system, which has beginning in the minute lymph capillaries. entire extent of the tubal mucosa is lined delicate network, which is more developed he cartilaginous than in the osseous part of tube and likewise is more abundant on the rior wall than on the other walls of the tube , 17). The lymph vessels course in mark- different directions in the tunica propria, in the submucosa they run mostly in the itudinal direction of the tube, uniting with other in an elongated network.[34] At the l of the isthmus of the tube the lymph chan- become less tubular in shape and tend to me widened sinusoid spaces.[24] Within this cate network lymphoid nodules are to be nd rather commonly in the tunica propria.[21] lier observers seemed to mention only a phoid nodule in the pharyngeal end of the , which they called the tubal tonsil (Gerlach) the noduli lymphatici tubarii (Spalteholz). wever, in people with good hearing, according Polvogt and Babb,[21] lymph nodes may be rved in all parts of the tube, either deep in tunica propria or on the surface of the lining mbrane. This tissue is found more frequently young persons, although it may be found in lts, even those of advanced age (65 years).

he collecting trunks from these tubal capil- es pass in a general posterior direction from posterior medial surface of the tube to either retropharyngeal nodes medially or the deep ical nodes laterally. Four routes of drainage be distinguished [35] in figure 17: the retro- ryngeal, the anterior, the posterior and the stympanic. The retropharyngeal route (*R* in 17) consists of one or two trunks issuing n the pharyngeal portion of the tube and sing downward and posteriorly to the lateral retropharyngeal nodes. The anterior route (*A* in fig. 17) is composed of two or three trunks issuing from the medial end of the pharyngeal portion of the tube. These vessels, being anterior to the styloid process, run backward along the tonsillar or the ascending palatine and external maxillary arteries to the anterior subdigastric node of the deep cervical chain. The posterior route (*P* in figure 17) consists of two or three trunks issuing from the posterior wall and inferior border of the tube. These trunks course posterior to the styloid process, one in front of the carotid sheath (but behind the external carotid artery) and the other behind it, to the subdigastric and upper deep lateral cervical nodes, respectively. The transtympanic route (*T* in fig. 17) consists of the continuity of the network of the osseous portion of the tube with the network of the tympanic cavity. The subsequent course is through the tympanic membrane, along the external auditory canal to end in the suprafascial or subfascial parotid nodes and the upper deep lateral cervical nodes (via the retroaural-sternomastoid route). This transtympanic route is difficult of demonstration, as shown by Arnould's demonstration of its existence in only 5 of 20 subjects.

Such a distribution of lymphoid vessels and tissue seems to substantiate the clinical findings of a greater frequency of lymphoid inflammation, acute inflammation, ulcerations, tuberculosis, adhesions and scars in the pharyngeal end of the tube, in contrast to the prevalence of osteitis, caries and necrosis with formation of granulation tissue in the osseous portion of the tube.[36]

NERVE SUPPLY

The sensory and the vasomotor nerve supply of the eustachian tube, along with the motor supply of the muscle fibers opening the tube, are to be considered here.

The sensory nerve supply of the tube is from the glossopharyngeal nerve (fig. 18) via the tympanic branch from the inferior (petrous) ganglion (fig. 14) through the tympanic plexus in the middle ear and thence to the tube. Clinical evidence in man that the tube is supplied afferently only by the glossopharyngeal nerve has been given by Erickson,[37] who found complete anesthesia of the tube following an intracranial section of the glossopharyngeal nerve for paroxysmal neuralgia. Definite evidence that the glossopharyngeal nerve (pharyngeal

Miyazaki, H.: The Finer Distribution of the ph Vessels of the Auricle, of the External Auditory tus, of the Eustachian Tube and the Pharyngeal il, Folia anat. japon. **11**:241-246 (June) 1933.

Rouvière, H.: Anatomy of the Human Lymphatic em, translated by M. J. Tobias, Ann Arbor, Mich., ards Brothers, Inc., 1938.

36. Halsted, T. H.: Pathology and Surgery of the Eustachian Tube, Arch. Otolaryng. **4**:189-195 (Sept.) 1926.

37. Erickson, T. C.: Paroxysmal Neuralgia of the Tympanic Branch of the Glossopharyngeal Nerve, Canad. M. A. J. **33**:647-650 (Dec.) 1935.

branches) supplies the pharyngeal orifice of the tube, the tubal elevation, the salpingopharyngeal fold and the salpingopalatine fold was first presented by Dandy [38] in 1927. In the 3 cases of the two most detailed studies of the innervation of the pharynx, by Dandy [38] and by Stookey,[39] following intracranial section of the glossopharyngeal nerve, the orifice of the tube is included in the area of supply. When one analyzes the subsequent observations of other surgeons, the general impression is reached that the tubal orifice is also entirely innervated by the trigeminal nerve in 50 per cent of the cases. This fact may be explained (without experimental proof) by the pharyngeal nerve supply from the sphenopalatine ganglion. Many of the branches of the pharyngeal nerve are tubal ones and descend directly to the entire pharyngeal portion of the tube. Whether the tube also may have trigeminal innervation is not definitely known for only one surgeon [37] has tested tubal sensitivity; in that study it proved to be entirely supplied by the glossopharyngeal nerve.

The vasomotor innervation of the tube comes from the sympathetic plexus on the internal carotid artery. Wolff [24] figures and describes it as a branch of the caroticotympanic plexus entering approximately half way up the tube which runs toward the middle ear along the posterior medial wall. It, like the glossopharyngeal nerve, is accompanied by an arteriole and two or more venules. No mention is made as to how the rest of the tube receives its sympathetic innervation.

The motor nerve supply to the tensor palati muscle, which alone opens the tube, is from the trigeminal nerve. As the mandibular branch of the fifth nerve leaves the foramen ovale it gives off a branch to the otic ganglion, thus transmitting the motor fibers which reach the muscle by way of the tensor palati branch of the otic ganglion (fig. 14). All of the closely

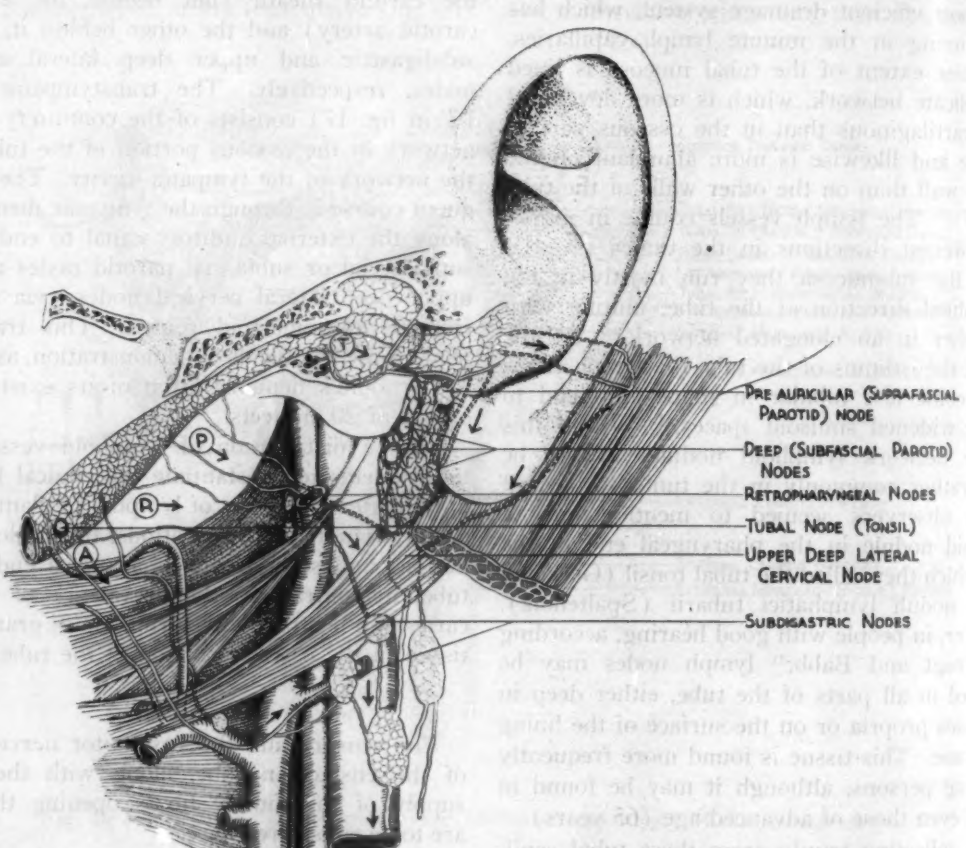


Fig. 17.—A drawing of the lateral view of the lymph drainage of the eustachian tube, based on anatomic descriptions in the literature. *A* indicates the anterior drainage route; *R*, the retropharyngeal route; *P*, the posterior route, and *T*, the transtympanic drainage route.

38. Dandy, W. E.: Glossopharyngeal Neuralgia (Tic Douloureux): Its Diagnosis and Treatment, Arch. Surg. **15**: 198-214 (Aug.) 1927.

39. Stookey, B.: Glossopharyngeal Neuralgia: Surgical Treatment, with Remarks on the Distribution of the Glossopharyngeal Nerve, Arch. Neurol. & Psychiat. **20**:702-719 (Oct.) 1928.

d muscles, such as the salpingopharyngeus, levator palati and the superior constrictor, innervated by the pharyngeal branch of the us nerve.

ROENTGEN VISUALIZATION OF THE TUBE

The study of the eustachian tube in the living bject was confined mostly to examination of pharyngeal orifice and probing of the tube til 1927, when Spielberg [7] first studied the e by iodized sesame oil and roentgenograms. s account gives measurements of the length, height and the angulation of the normal tube, ich correspond to those of the cadaver. He

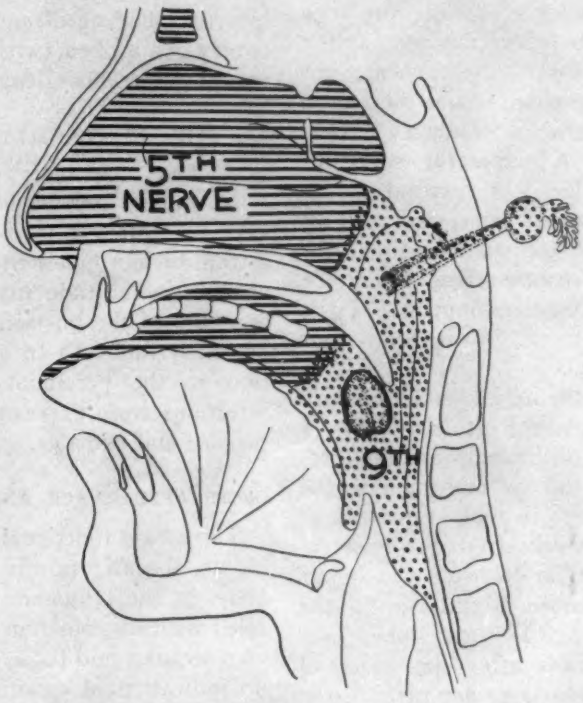


Fig. 18.—A diagram of the sensory nerve supply of the eustachian tube, the middle ear, the mastoid cells d the pharynx. The nerve is the glossopharyngeal nerve.

inted out the use of the method to visualize e presence and the sites of strictures or stenoses thout danger to the patient. He did, however, dicate the possible retention of the iodized l for as long as one week after its injection in lated or atrophic tubes, in contrast to the veral minutes required for emptying by normal lia or swallowing. Rees-Jones and McGibbon [40] ve found iodized oil too viscid for satisfactory e and have used pyelectan (a solution of the odium salt of 3,5-diiodo-4-pyridoxyl-N-methyl-6-dicarboxylic acid) to visualize 34 tubes with ockage in so-called aviation pressure deafness.

40. Rees-Jones, G. F., and McGibbon, J. E. G.: adiological Visualization of Eustachian Tube, Lancet :660-662 (Nov. 29) 1941.

Their studies showed that the blockage was in the central portion of the cartilaginous part in 91 per cent of the affected tubes, at the pharyngeal orifice in 3 per cent and at the isthmus in 7 per cent. Their technic of visualization is clearly stated and has been used with obvious success in the diagnosis of deafness in the Royal Air Force.

MALFORMATIONS

Descriptions of the developmental defects of the eustachian tubes are meager. Greig [41] described a skull in which the tympanic element of each temporal bone was absent, resulting in a deficiency of the osseous portion of the tube. A similar case was reported by Ranke [42] in 1889. Krukover [43] reported a congenital bilateral malformation of the ear in which there were defects of the eustachian tube.

TUMORS OF THE EUSTACHIAN TUBE

Since 1888 there have been 30 cases of primary tumor of the eustachian tube, and all but 1 of

41. Greig, D. M.: Congenital Absence of the Tympanic Element of Both Temporal Bones, J. Laryng. & Otol. **42**:309-314 (May) 1927.

42. von Ranke, H.: Case of Malformation of the Ear, Sitsungsb. d. Gesellsch. f. Morphol. u. Physiol. in München **5**:68, 1889-1890.

43. Krukover, I., cited by Meltzer, P.: Anatomy and Physiology of the Ear, Arch. Otolaryng. **7**:167-183 (Feb.) 1928.

these have been reported since the advent of instruments which enable one to visualize directly the orifice of the eustachian tube [44] or to see it indirectly, as by the nasopharyngoscope [45] or by posterior rhinoscopy. In 1912 Proctor [46] reported the first case in the American literature of a highly malignant carcinoma. In 1940 Shumaker [47] recorded the first cure (three years) of a patient with primary carcinoma of the tube. Lawson [48] has summarized the existent literature on primary carcinoma of the eustachian tube and has found that "most of the neoplasms with this diagnosis arose from the cartilaginous portion of the tube." However, tumors of the tube do arise in all its portions, and only a cautious suspicion of a tumor's possible presence will lead to its diagnosis before autopsy, as the middle and outer portions of the tube are not easily accessible to observation. As to the region near the pharyngeal orifice, Shumaker [47] cautioned that in cases of unilateral secretory catarrhal otitis which does not respond to the usual methods of treatment after two to four weeks, a tumor of the eustachian tube should be suspected, and weekly examinations should be made until either the condition improves or the cause for the failure to do so is known or appears at the tubal orifice.

Deafness seems to be the initial symptom, with tinnitus frequently complained of, occasionally a full feeling in the ear or intermittent pain referred to the depths of the ear (referable to the glossopharyngeal nerve). It took a patient of Shumaker's [47] thirteen weeks from the onset of symptoms to present visible evidence of a tumor at the orifice of the tube. Metastasis to the cervical lymph nodes (fig. 17) may take place as early as three months after the onset of symptoms.[49] Distant metastases are rare. Local infiltration extending far about the tube gives rise to a train of new symptoms indicating the hopelessness of the situation. Such symptoms include reduced visual acuity, diplopia and paralysis of ocular muscles, due to extension through the basal foramens, the orbital fissure or the jugular foramen.[48]

FOREIGN BODIES IN THE TUBE

Owing to the expansion of the eustachian tube in both directions from the isthmus, two approaches for the removal of foreign bodies in the tube are possible. Each approach has been successfully used in the only 2 cases recorded in the literature. Hastings [50] removed a piece of a gold bougie from the distal half of the tube at the time of a radical mastoid operation. Mangiaracina devised special grasping forceps to be introduced into the pharyngeal end of the tube for the recovery of a broken, twisted wire applicator, which occupied the entire length of the tube.

THE EUSTACHIAN TUBE OF THE NEWBORN

Nowadays the newborn infant travels wherever its mother goes, be it on an airliner or on a train or in a motor over high mountains. Sudden changes of altitude make an exposition of the anatomy of the eustachian tube of the newborn of timely interest. In addition there is the relation to the perennial problem of pharyngeal infections from exposure to various and sundry peoples and climates.

GROSS APPEARANCE AND CLINICAL PHYSIOLOGY

The tube is functional soon after birth. According to Bryant,[8] no air enters the tube immediately, as the tympanic cavity of the newborn is filled with myxomatous tissue and detritus. Our own sections and those of others,[24] however, give no indication of detritus or myxomatous tissue in the tympanic cavity or the tube. Nevertheless, Bryant expressed the belief that the action of the tensors produced by suction rapidly stimulates the opening of the tube.

The pharyngeal orifice is close to the vault of the nasopharynx, owing to the low roof of the elongated anteroposterior axis (fig. 19). The orifice is a deep funnel-shaped pit directed anteromedially, which has a shallow groove curving from it posteroinferiorly for several millimeters between the horizontal prominence of the levator palati muscle anteriorly and the elevation of the tubal cartilage posteriorly. The pit of the orifice and its increasingly shallower tail-like groove

44. Yankauer, S.: A Speculum for the Direct Examination and Treatment of the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **20**:421-422, 1911.

45. Holmes, E. M.: Examination and Treatment of the Eustachian Tube by the Aid of the Nasopharyngoscope, Ann. Otol., Rhin. & Laryng. **20**:29-40 and 511-530, 1911.

46. Proctor, P. C.: Two Cases of New Growth Involving the Eustachian Tube, Boston M. & S. J. **166**:96, 1912.

47. Shumaker, P. W.: Primary Carcinoma in the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **49**: 1038-1043 (Dec.) 1940.

48. Lawson, L. J.: Primary Carcinoma of the Eustachian Tube: A Study of the Evidence of Its Occurrence, Ann. Otol., Rhin. & Laryng. **52**:377-390 (June) 1943.

49. Brownell, D. H.: Primary Carcinoma of the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **49**:551-555 (June) 1940.

50. Hastings, H.: A Piece of Gold Bougie (Two Years in Situ) Removed from the Distal Half of Eustachian Tube During a Radical Mastoid Operation, Ann. Otol., Rhin. & Laryng. **46**:248-251 (March) 1937.

51. Mangiaracina, A.: Foreign Bodies in the Eustachian Tube, Arch. Otolaryng. **32**:517-519 (Sept.) 1940.

emble a comma in shape. The pit lies on a izontal line directed through the hard palate ghly half way between the nasal septum and posterior pharyngeal wall. Numerically this sually 2 mm. inferior to the roof, 6 mm. pos- or to the base of the nasal septum and 7 to nm. anterior to the posterior pharyngeal wall. us the pharyngeal orifice of the newborn is in nasopharynx are the prominences of the cartilaginous extremities of the Eustachian tubes which stand out sharply from the side walls." The elevation or torus made by the tubal cartilage (fig. 19) lies as a curved hood over the superior and posterior margins of the orifice. As this posterior fold fades out inferiorly it does not cover a conspicuous salpingopharyngeal muscle

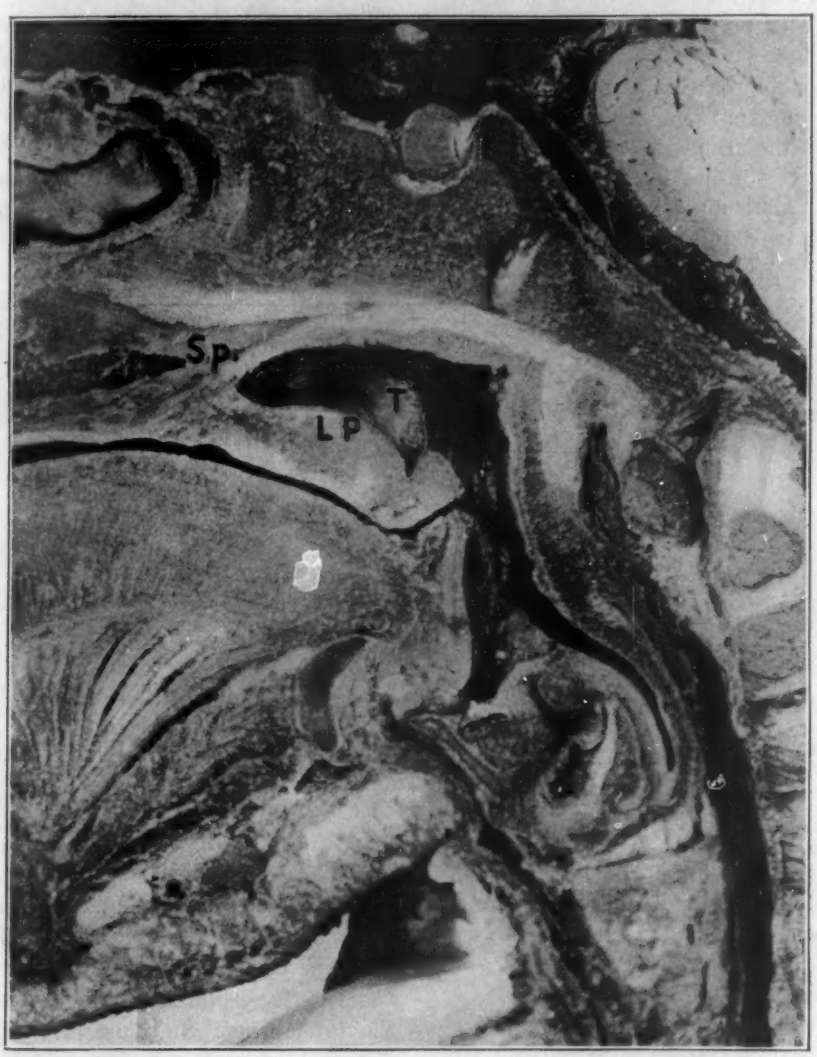


Fig. 19.—A photograph of the pharyngeal orifice of the eustachian tube in the embalmed body of a newborn nt. *LP*, indicates the levator palati eminence; *SP*, the posterior end of the nasal septum; *T*, the torus ation of the tubal cartilage with the orifice of the eustachian tube anteriorly and the fossa of Rosenmüller eriorly.

tively the same position to the nasal septum the posterior nasal wall as in the adult, ough found much nearer the roof of the ity.

We agree with Braislin [52] that "the most conuous landmarks in the lateral wall of the as it does in the adult, for this muscle is represented by only a few strands of muscle fibers. A true anterior fold does not exist in the infant. However, the levator torus is much accentuated and sweeps directly medially in a horizontal plane. All this is in marked contrast to the adult condition, in which growth has stretched the folds into more vertical positions.

In dimensions the infant's tube is one-half the length of that of the adult, averaging 18 mm.,

52. Braislin, W. C.: A Study of Some Casts of the antile Pharynx with Special Reference to the Eustan Tube, Ann. Otol., Rhin. & Laryng. **19**:36-49 arch) 1910.

although Kerrison [53] gave the length as 14 mm. and Bryant [8] as 20 mm. The pharyngeal portion of the tube maintains in length a 2 to 1 ratio to the osseous portion, as in the adult. The lumen of the pharyngeal portion is about the same width (1 mm.) as that of the adult, although the height is nearly one-half that of the adult for comparable sections (figs. 2 and 20). The direction of the tube in the newborn varies from a horizontal plane (fig. 20) to one of about 10 degrees of inclination.

No angulation of the tube marks the isthmus (fig. 21), only a narrowing in all diameters of the tube. As in the adult, the isthmus is located at the junction of the middle and lateral thirds.

The tensor palati muscle is well formed at birth and has an extensive attachment to the well defined lateral fibrous membrane (salpingopharyngeal fascia) and to the hook of the tubal cartilage (fig. 22). It is the function of this muscle to pull on the salpingopharyngeal fascia, which supports the underlying mucosa, thereby opening the closed or resting tubal lumen. Since the tensor palati muscle is brought into contraction during swallowing, it is the custom on commercial airliners to induce regular and frequent swallowing by having the baby suck the nippled water bottle. This act, or that of nursing, causes the newborn infant to increase the normal (awake) rate of swallowing of five times a minute to twenty times a minute. Equally important, as with adults, the sleeping infant must be awakened in descent of sleeper planes, for the swallowing rate is only once in every three minutes when the infant is asleep.

53. Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1923.

MICROSCOPIC APPEARANCES

In order to correlate anatomic and physiolo facts with certain clinical observations and ap cations, detailed microscopic appearances of va ous portions of the infant tube are here describ

Pharyngeal End.—The pharyngeal orifice the tube deserves major emphasis as it is t site of greatest assault by mechanical facto infection and therapeutic maneuvers. Here t thick pseudostratified epithelium of the phary geal wall continues over the elevated margin the orifice of the tube, dips into the funnel a then gradually thins as it nears the tube prop (fig. 23). Tiny pits over the surface of the tor admit to the openings of the large number small compound tubuloalveolar (mucserou

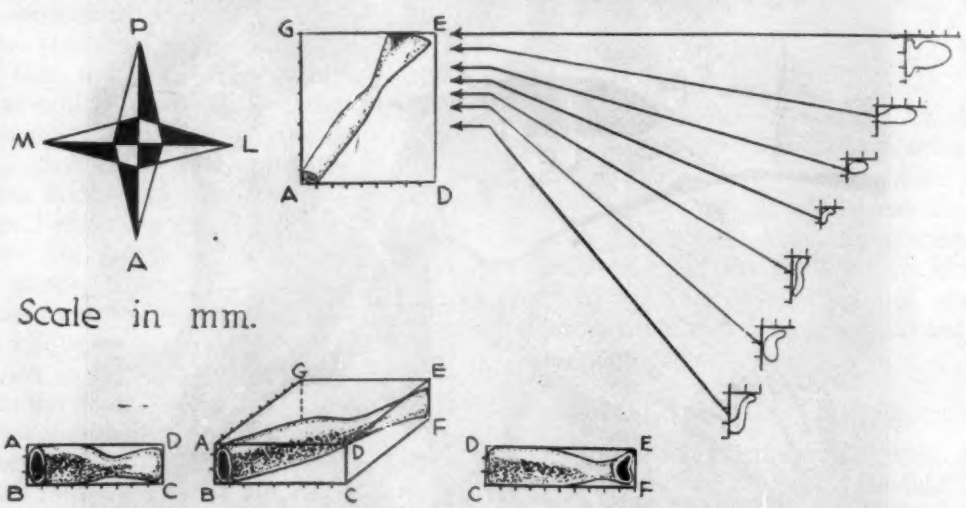


Fig. 20.—A drawing of plane and three-dimensional views of the newborn infant's eustachian tube, w outlines of cross sections of the lumens of the tube at various levels. *A G E D* is the superior view; *B A D* the anterior view; *C D E F*, the lateral view. All figures are drawn to a millimeter scale, as indicat

glands which lie embedded in the loose tuni propria. These glands are several clusters de over the torus and in the funnel and are sep rated by loose fibrous tissue. At the entrance the tube proper the clusters lose their identi as the glands are packed more tightly, one-half thick as in the funnel and one-sixth as thi as over the torus.

The vascularity of the orifice of the tube well shown in figure 24 *A* and *B*, where t subepithelial and glandular networks stand o distinctly. Clearly does this bring out the enc mous potentialities of irritants to bring abo engorgement in such an extensive vascular b and indeed to cause edema in the loose fibro tunica propria supporting this vascular netwo

Cartilaginous Portion.—Here the mucosa sho low longitudinal folds of pseudostratified e thelium except in the superior portion of the tu

jacent to the hook of cartilage, where there are folds (fig. 26 *A* and *B*), and the epithelium anges to a simple columnar type. The folds the medial wall of the tube are two to three nes the height of those on the lateral wall. ilia are found on the surface of both of these with the lateral side, which is denser and poorer in cells. No tunica propria exists beneath the simple epithelium at the cartilage hook, for it rests directly on the perichondrium.

Numerous mucoserous glands occur in the medial wall, a lesser number in the lateral wall;

Fig. 21.—A photograph of a horizontal celloidin section of a newborn infant, made through the eustachian bes. *ET* is a section through the superior portion of the eustachian tube connecting to the middle ear (ME); is the pharynx. The tensor palati muscle is seen arising from the entire length of the lateral lamina of the bal cartilage. Lateral to the right tube are seen the mandibular nerve, the middle meningeal artery, the terygoid muscle and the pterygoid plexus.

pes of epithelium. Wolff [24] claimed to have found cilia only on the roof near the pharyngeal end.

The tunica propria is thicker on the medial side, being loose and very cellular in comparison the glands gradually disappear entirely about halfway up the cartilaginous portion of the tube. The openings of these glands may be traced to the depths of the crypts between the high folds of mucosa (fig. 27).

There is a subepithelial collection of lymphoid tissue, known as the tubal tonsil, about 3 mm. lateral to the pharyngeal orifice on the medial wall. This tonsil is located well superior in the tube, while that figured by Wolff [24] lies well inferior although still in the medial wall. Urbantschitsch [18] mentioned the frequency with which countless lymphoid nodules are found in the middle part of the cartilaginous tube in the child. Such tubal tonsils cause a raised hillock of the mucosa, giving a clue to their importance in tubal occlusion by enlargement. Polvogt and Babb [21] concur in the common impression that

Osseous Portion.—The pseudostratified e thelium of the mucosa in the osseous portio covered with cilia. Only at the lateral por of the superior wall is the epithelium reduce cell layers, and there it rests directly on the p chondrium, thus marking the continuation of axis of the tube from more medial portio The low folds of the isthmus continue into portion only in the inferomedial corner (26 *D*) and are increased in height. Beneath th high folds is found a very vascular tunica prop (fig. 25 *C*) made up of loose coarse fibers c tinuous with the fibrous periosteum. The loo

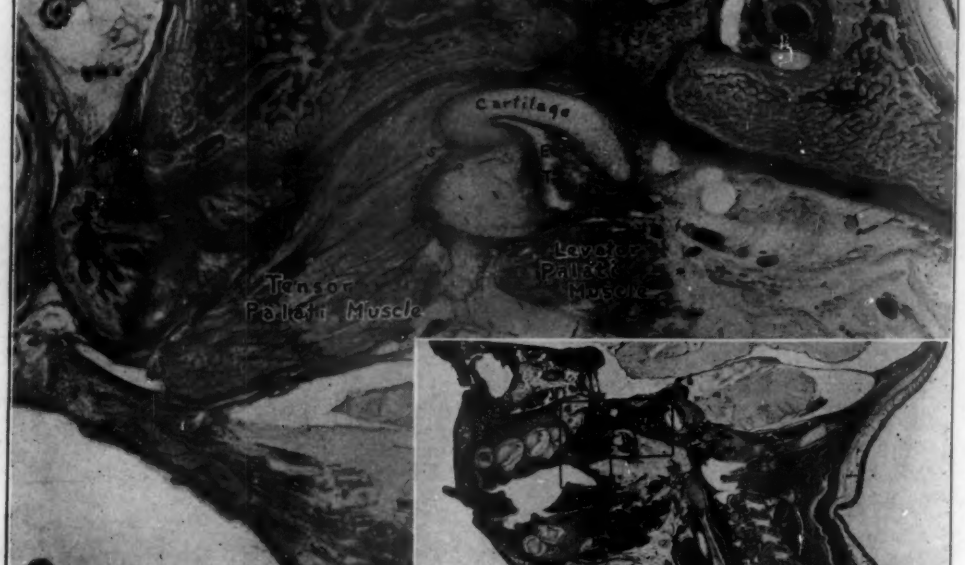


Fig. 22.—A photograph of a sagittal celloidin section of a newborn infant, made through the pharyn end of the eustachian tube, showing the muscle relations to the tube (*ET*), the cartilage and the salpin pharyngeal fascia (*S-P* fascia). The inset shows the position of the photographed area.

these are more frequently seen in young persons, although they may be found even as late as 65 years.

Isthmus.—The epithelium of the isthmus is everywhere pseudostratified, although the thickness is reduced adjacent to the cartilage superiorly. Cilia are found throughout. Low folds of mucosa are seen on the floor and on the inferior portions of the medial and lateral walls (fig. 26 *C*). The tunica propria is thin and acellular, contains no glands and is fused to the perichondrium at the superior end of the lumen next to the cartilage.

ness of the tunica propria and the lack of comp bone adjacent to the mucoperiosteum offer substantial barrier to the spread of infection i the spongy bone at the inferomedial corner of tube. In this same corner numerous branc of the caroticotympanic nerve plexus are se Beneath the superolateral epithelium, de fibrous tissue characterizes the tunica propria

Tympanic Ostium.—The mucosa is similar that found elsewhere in the tube (pseudostrati ciliated). Only along the roof adjacent to cartilage does the epithelium become a t layered columnar type resting directly on

perichondrium. Low folds are observed in medial part of the floor (fig. 26 *E*). The se, vascular tunica propria (fig. 25 *D*) forms hin layer beneath the epithelium except where ests directly on the perichondrium.

Two evaginations occasionally occur at this end the tube. The subtubal air cells may evaginate m the inferomedial angle (fig. 26 *E*), although y are more usually found evaginating from the ddle ear—in 90 per cent of infants.[25] The pre-otid air cells are seen to bud off from the eromedial corner of the tube into the bone ween the tensor tympani muscle and the

THE EUSTACHIAN TUBE IN MILITARY PURSUITS

AVIATION

Aviation medicine is concerned with those conditions which are primarily physiologic and subclinical. Malfunction must be prevented or set aright and disease conditions must be sought out, since they are for the most part disqualifying in military if not in commercial flying.

Since the earliest days of man's adventuring into air, in 1783, the ear has demanded painful attention. As a result of the intervening years

Fig. 23.—Photograph of a horizontal celloidin section of the newborn infant's eustachian tube, showing its lationships. The pterygoid venous plexus and the mandibular nerve are shown intervening between the sor palati and the internal pterygoid muscle.

rotid artery (fig. 26 *E*). Bast and Forester [25] ve observed them in 40 per cent of infants. heir origin is not always from the eustachian be but may be from the middle ear cavity or m the subtubal cells. These air cells in the mporal bone are lined with the same type of thelium as the eustachian tube and likewise ve a loose vascular tunica propria buffering em from the periosteum and the compact bone. addition, emphasis should be placed on the t that there is a thin plate of compact bone tween them and the carotid canal, offering finite resistance to spread of infection.

of search, the eustachian tube has come to be known as the offending structure. It seems to act as a flutter valve connection between the pharynx and the middle ear to equilibrate alterations in pressure of the environment.

Level Flying.—On level ground or in level flying the slight variations of pressure within and without the middle ear are adequately adjusted by the normal swallowing rates of man and the resultant contractions of the tensor palati muscles to open the tubes. An adult usually swallows once a minute while awake and once in five minutes while asleep. This rate is accelerated

to one swallow every one to thirty seconds while chewing.

Ascent.—On ascending into the air from the take-off, the decreasing pressure of the atmosphere allows a slight outward bulging of the tympanic membrane and a slight sense of fulness in the ears when the distance from the earth is 100 feet (30.5 meters) (3 to 5 mm. of mercury differential), provided no swallowing has taken place (fig. 28). When the ascent has been ma to 500 feet (10 to 15 mm. of mercury differenti without swallowing, the hearing becomes dist and of less intensity and is then sudde relieved as the increased air pressure in the m dle ear forces an automatic opening of the tub with its attendant "click". Thus on continue ascent the increased air pressure within the mi dle ear forces successive openings of the tube i

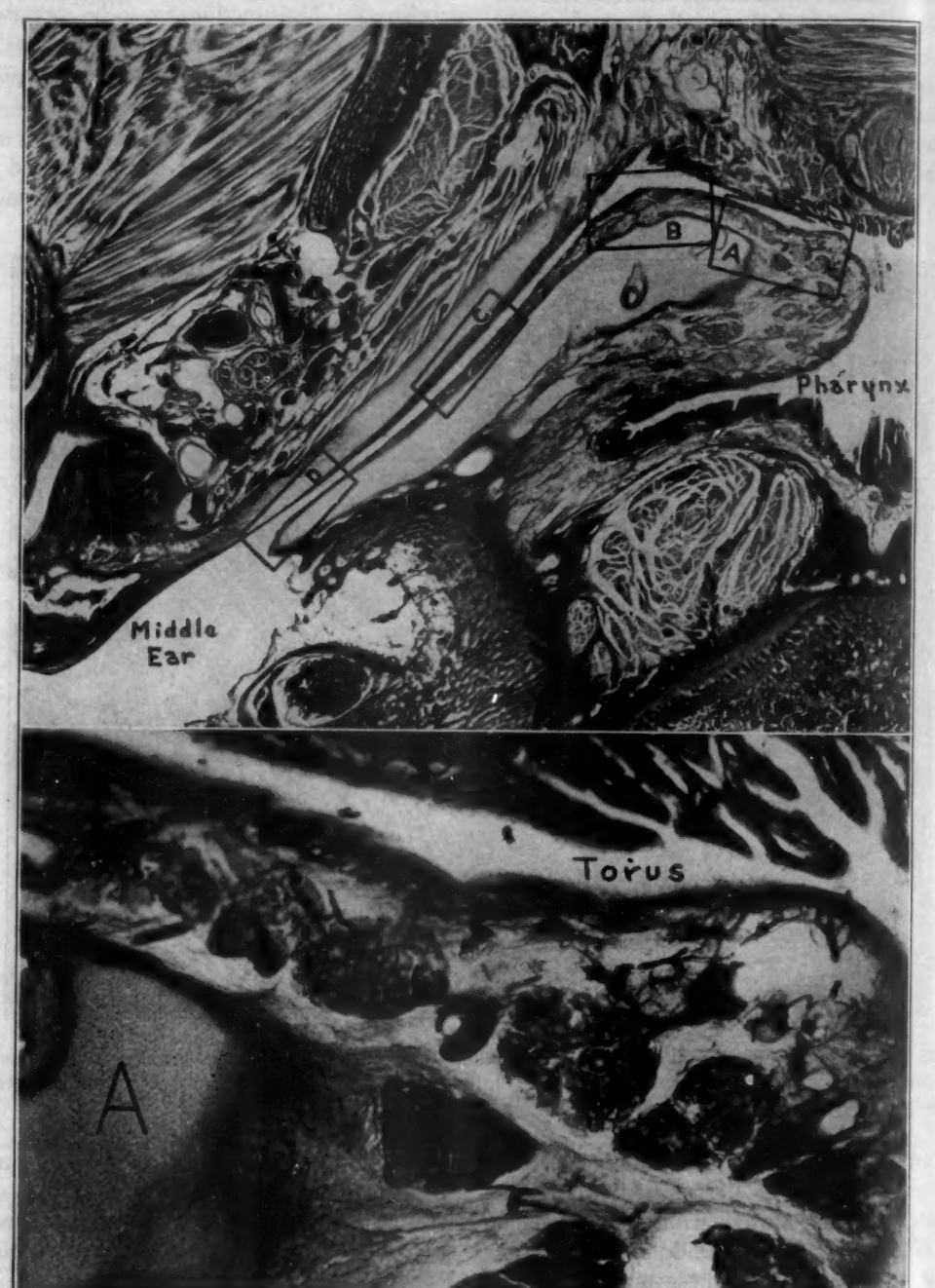


Fig. 24.—Photographs of the newborn infant's eustachian tube showing the vascular patterns of the tuni propria. The top photograph is identical with figures 28 and 30 and gives the key to the magnifications *A* (region of torus) and (in fig. 25) of *B* (pharyngeal orifice), *C* (cartilaginous portion) and *D* (tympanic orifice

atively decreasing frequencies as is shown in ure 28, and all of these openings are entirely dependent of the openings produced by normal allowing. With these two mechanisms work- simultaneously to open the tube, little diffi- lty is experienced by aviators in ascent, pro- 100,000 feet per minute without giving a thought to his ears. This is substantiated by other experiments with the low pressure chamber, which "have proved that even in sudden decreases of barometric pressure there is almost a complete absence of any ear symptoms, which suggests

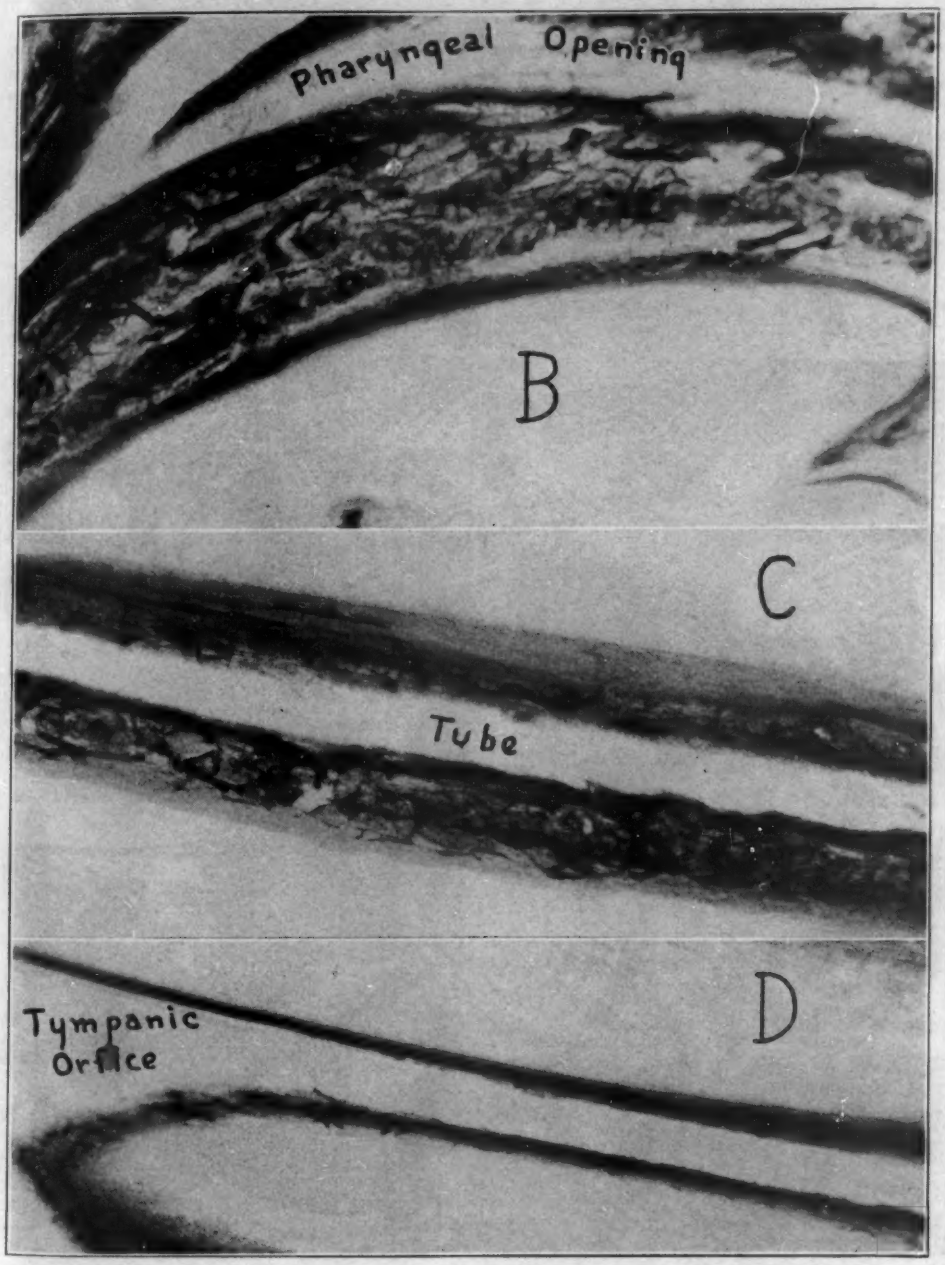


Fig. 25.—Photographs of horizontal celloidin sections of the newborn infant's eustachian tube showing ascular patterns in the tunica propria. *B*, *C* and *D* are located by the key at the top of figure 24.

ided infection or previous trauma does not pre- lude opening of the tube. Whaley [54] has "gone p" in the decompression chamber at the rate of that the expanding gas in the middle ear immediately and easily finds escape through the tube." [55] If for some reason the tube remains closed until there has been an ascent of 1,000 feet (15 to 30

54. Whaley, J. B.: Problems of Aviation Medicine elating to Ear, Nose and Throat, Arch. Otolaryng. 6:438-442 (Sept.) 1942.

55. Carson, L. D.: Otolaryngological Aspects of Aviation, Laryngoscope **52**:704-716 (Sept.) 1942.

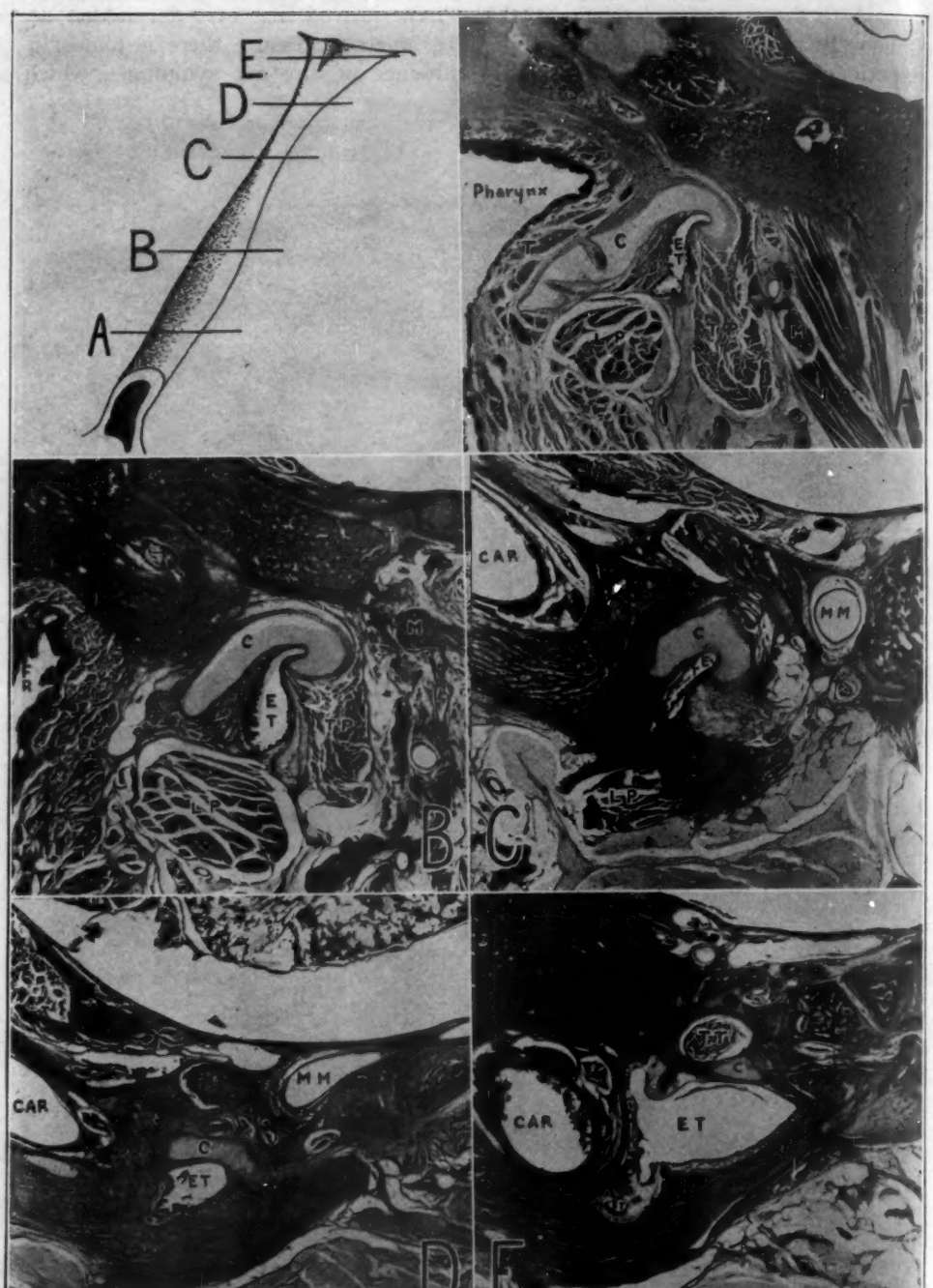


Fig. 26.—Photographs of frontal celloidin sections at various levels of a newborn infant's eustachian tu
The levels are indicated on the inset drawing. *C* is the cartilage; *Car*, the internal carotid artery; *ET*, t
lumen of the eustachian tube; *FR*, the fossa of Rosenmüller; *IP*, the internal pterygoid muscle; *LP*, the levat
palati muscle; *M*, the mandibular nerve; *MM*, the middle meningeal artery; *ST*, a subtubal air cell; *T*, t
torus; *TP*, the tensor palati muscle; *TT*, the tensor tympani muscle.

n. of mercury differential), tinnitus develops h sensations variously described as hissing, ring, snapping and crackling sounds in the s, also vertigo and sometimes pain.

Descent.—Descents from altitude almost al- ys require some conscious maneuver to venti- the middle ear in order to equalize the pres- pharynx to initiate the opening [56] either by swallowing or by contracting the abdominal muscles. "In spite of almost universal autoinflation of the ears among our navy pilots, 'head cold' or no 'head cold' and disregarding sore throats, the actual incidence of acute otitis media is found to be no higher among flyers than in the case of

Fig. 27.—A photomicrograph (× 160) of the mucous membrane of the cartilaginous portion of an infant's tachian tube, showing a pit into which the duct of a mucous gland is opening.

re there with the increasing barometric pres- re. Swallowing, yawning, sneezing are the tural movements opening the tubes. Tensing d relaxing the tensor muscles by moving the ndible will often suffice, but there are a num- r of persons who must resort to closing the trils and exerting enough pressure in the mere groundlings." [55] In the rapid descents of dive bombing the continuous yell or the continuous swallowing maneuver is used. Here the tube probably remains open while considerable gas enters the middle ear.[56]

56. Poppen, J. R.: The Ear in Flying, Tr. Am. Otol. Soc. **31**:282-291, 1941.

It is quite common for passengers in commercial air liners to complain that the pressure will not equalize for several hours after descent. This is because the 21% of oxygen must be absorbed down to 12 to 15% in the capillaries. Happily, there is a simple solution which requires very little attention. If the air introduced is alveolar air, it will approximate very closely the composition of that in the capillaries. Thus, if the ear is inflated at the end of expiration following moderate breath-holding, the period of recovery will be very materially shortened. This simple procedure is offered as a therapeutic means to those who have experienced protracted delay in reestablishing normal pressure in the middle ear.[56]

[Occasionally there are instances] in which every possible maneuver fails to accomplish equalization of pressure—and these unfortunates are usually pitiable objects. When one of these pilots walks into the office, the diagnosis can be made from the facies and a casual glance at the clothing. The face registers acute misery, the eyes are reddened and lacrimose. He hasn't bothered to remove all of his flight clothing before seeking relief.[56]

Inefficiency of the eustachian tubes in opening has brought on a whole series of symptoms and signs, which have acquired in the recent past more accurate anatomic and physiologic explanations. In the subclinical range of symptoms are those experienced in commercial aviation. Here the limits of descent are between 300 and 500 feet (90 and 150 meters) per minute, which give no or only slight discomfort when the occupants of the plane are awake and swallowing the normal rate of once per minute (fig. 29).

However, the sleeping air traveler or unconscious or drugged soldier returning from the front by air transport is in danger at even these slow descents, for the rate of swallowing (tubal opening) while sleeping is only once five minutes. This slow rate of swallowing must be accelerated by waking and by drinking and chewing.

Descents in the range of 1,000 feet (300 meters) per minute, which sometimes occur in military aviation where air fields are nestled in valleys (fig. 29), give rise to moderate pain

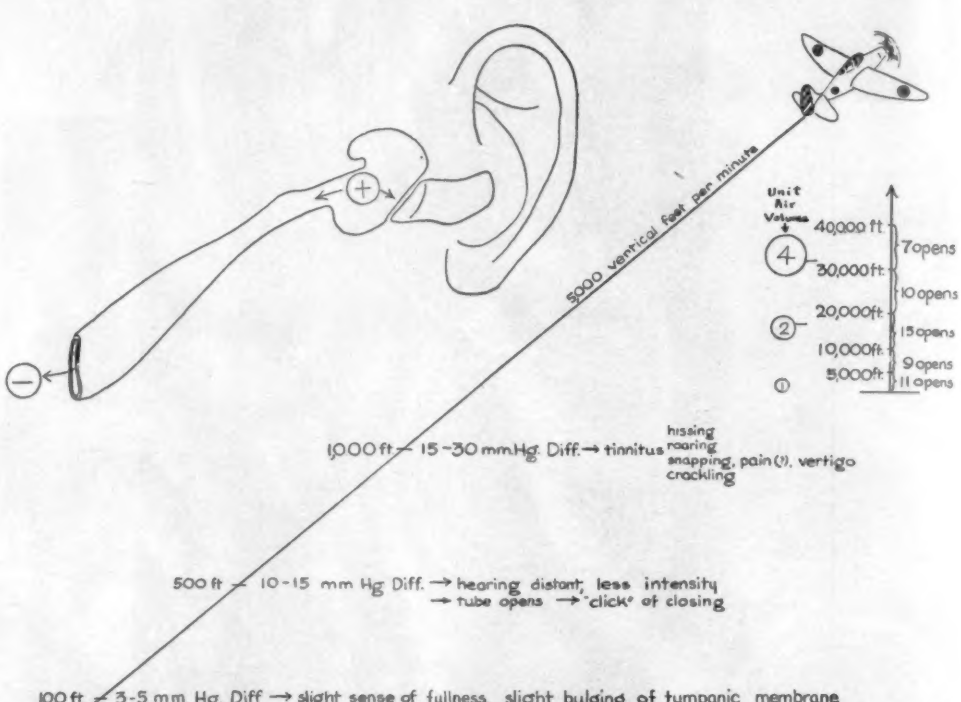

5000 vertical feet per minute
Unit Air Volume
40,000 ft.
7 opens
30,000 ft.
10 opens
20,000 ft.
15 opens
10,000 ft.
9 opens
5,000 ft.
11 opens
1,000 ft. — 15-30 mm. Hg. Diff. → tinnitus
hissing
roaring
snapping, pain (?), vertigo
crackling
500 ft. — 10-15 mm Hg Diff. → hearing distant; less intensity
→ tube opens → "click" of closing
100 ft. — 3-5 mm. Hg. Diff → slight sense of fullness, slight bulging of tympanic membrane

Fig. 28.—Physiology of ascent—a drawing to indicate the sensations experienced at various distances ascent when there is no swallowing or automatic opening of the eustachian tube. The inset below the airpl shows the number of automatic openings due to unit air expansion with measured ascent, while the ear tube inset suggests the mechanism of automatic tubal opening.

when man's normal swallowing rate is not co sciously accelerated. This may become unbe able and result in "locking" of the eustachi tubes, the clinical condition called "aero-ot media" in America after Armstrong[57] and "a ation pressure deafness" in England af McGibbon.[58]

Dive bombing descents up to 40,000 f (12,000 meters) per minute during tubal in

57. Armstrong, H. G.: Principles and Practice Aviation Medicine, Baltimore, Williams & Wilk Company, 1939.

58. McGibbon, J. E. G.: Aviation Pressure Deafn J. Laryng. & Otol. **57**:14-22 (Jan.) 1942.

ency bring out the complete picture of "aero-is media." "It has been repeatedly observed in fairly rapid descents from high altitudes first part of the dive causes no distress, reas acute distress will begin at between 00 and 20,000 feet (4,500 and 6,000 meters) at lower altitudes. The increasing density the air probably has something to do with ".[55] This increasing density of the air is in carefully shown by McGibbon's [58] figure 4, which the doubling and tripling of air pres-es on both the eardrum and the walls of the opharyngeal orifice are shown in comparing descent from 40,000 to 30,000 feet (12,000

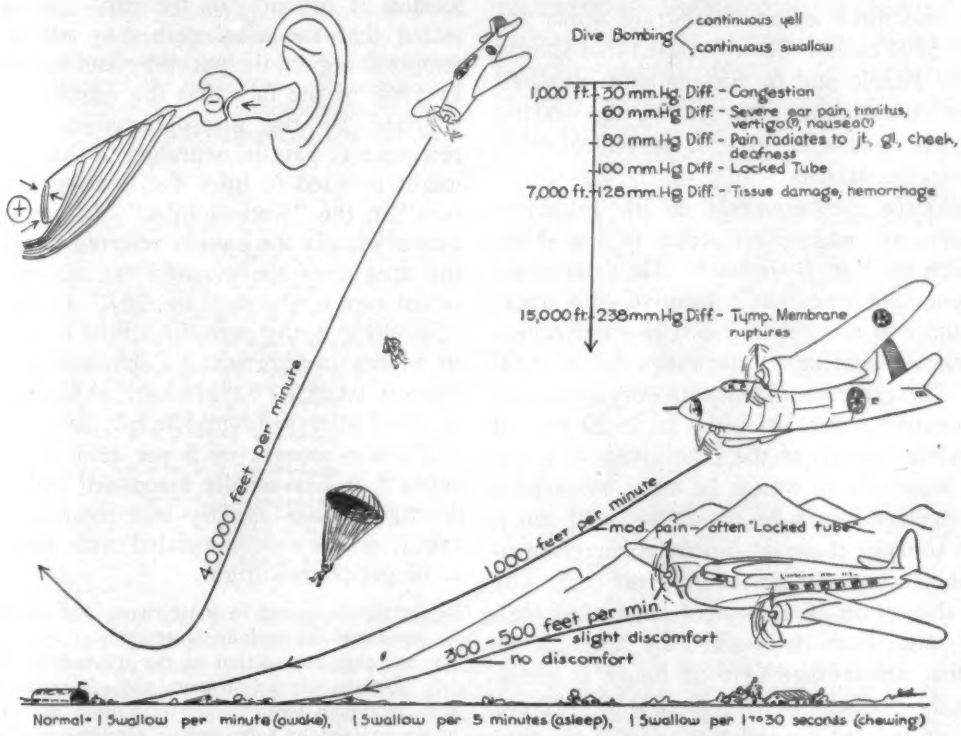

Dive Bombing
continuous yell
continuous swallow
1,000 ft.
30 mm.Hg Diff. - Congestion
60 mm.Hg Diff. - Severe ear pain, tinnitus, vertigo(?), nausea(?)
80 mm.Hg Diff. - Pain radiates to jt., gl., cheek, deafness
100 mm.Hg Diff. - Locked Tube
7,000 ft.
128 mm.Hg Diff. - Tissue damage, hemorrhage
15,000 ft.
238 mm.Hg Diff. - Tymp. Membrane ruptures
40,000 feet per minute
1,000 feet per minute
mod. pain - often "Locked tube"
300 - 500 feet per min.
slight discomfort
no discomfort
Normal - 1 Swallow per minute (awake), 1 Swallow per 5 minutes (asleep), 1 Swallow per 1 to 30 seconds (chewing)

Fig. 29.—Physiology of descent—a drawing indicating the sensations experienced during aerial descent in ansport planes (300 to 500 feet per minute), in combat planes making landing fields in mountainous valleys 000 feet per minute) and in dive bombers (40,000 feet per minute) under the normal automatic swallowing ndition of 1 per minute. The inset at the top at the right shows the symptoms often occurring at the rious foot descents and barometric pressure differentials. The inset at the top at the left suggests the echanism of the origin of symptoms of "acute aero-otitis media" or "locked tube."

9,000 meters) and the descents from 20,000 10,000 feet (6,000 to 3,000 meters) and 10,-0 to 0 feet. Armstrong's [57] data on the velopment of symptoms and signs during pid descents are recorded in the upper right-nd corner of figure 29.

The frequency of occurrence of these aural mptoms in the air forces during descent is dicated by McGibbon's [58] figure of 27 per nt of the total number of aircrew personnel.

Symptoms and Signs of Inefficiency of the ube.—(*a*) Symptoms: The outstanding single mptom of the "blocked eustachian tube" is deafness of the "conductive" type (10 to 30 decibels) for low tones (C to C_1 or 128 to 2048 double vibrations) which occurs in 94 per cent, possibly 100 per cent of cases, according to McGibbon,[58] or as a solitary symptom in 31 per cent of cases. Deafness is not only the most frequent symptom but the one which lasts the longest, having an average duration of sixteen days (four to twenty-eight days). Low tone deafness in these cases is due to one or the other or both of these concomitant developments: There is a marked retraction of the tympanic membrane, due to the difference in pressures on its two sides, which will limit the amplitude of membrane movement. As a result of this or as an independent reflex phenomenon (ultimately through the fifth nerve), the tensor tympani muscle may be shortened, resulting in increased rigidity of the ossicular chain and so impairment of the transmission of all low tones.[59] These clinical studies Loch [60] has substantiated

59. Wever, E G., and Bray, C. W.: The Tensor Tympani Muscle and Its Relation to Sound Conduction, Ann. Otol., Rhin. & Laryng. **46**:947-961 (Dec.) 1937.

60. Loch, W. E.: Effect of Experimentally Altered Air Pressure in Middle Ear on Hearing Acuity in Man,

(Footnote continued on next page)

by Valsalva's method of experimentally changing pressures. In the normal ears of 6 subjects he determined, by a Western Electric Company 1-A audiometer in a sound-proof room, that slight negative pressures caused an impairment of thresholds for frequencies above 5793 cycles per second and that as the degree of negative pressure was increased the thresholds for middle tones, and later those for low tones, also became impaired. With a marked degree of negative pressure there was usually impairment for all frequencies below 2048 of from 20 to 30 decibels, similar to that caused by a marked degree of positive pressure. Neither negative nor positive pressure had much effect on hearing acuity for 4096 and 5793 cycles. Loch [60] stated that similar studies by Bezold and by Siebenmann, although they used the tuning fork for testing hearing acuity, gave results in general agreement with those from his studies.

Quantitative measurements of the intratympanic pressure causing retraction of the drum were made by Van Dishoeck.[61] He determined by his pneumophone that a positive or a negative pressure of more than 2 cm. of water causes a decrease of hearing in the range below 2000 cycles. The presence of inflammatory processes gives negative values of —10 to —20 cm. of water, while catarrh of the tube gives —50 cm. values. Normally in winter he often observed a slight negative pressure (—2 to —10 cm.), while in summer these deviations occurred principally in persons who smoked a great deal. The rate of absorption of air out of the closed middle ear cavity was determined by him, for he stated that an average of four hours is necessary for the pressure to drop from 0 to —20 cm. If nitrogen is used for inflation, double the time is necessary. He also found that the average resistance of the orifice of the tube to opening was about 30 cm. of water in normal test subjects. This corresponds fairly closely to the Mao and Hughson [62] figures on children and adolescents of the Pennsylvania School for the Deaf. They found an opening pressure of about 40 cm. for boys and 39 cm. for girls in groups of young persons from 9 to 21 years of age.

Pain occurs in 61 per cent of obstructed tu and as a solitary symptom in 6 per cent.[58] may be

> . . . mild, or so severe as to cause an airman break off a dog-fight and even to make him faint. .
>
> Experiments seem to show that the main factor the pain in the ear lies not in the bulging of the dr head but (as in the case of the sinuses where no dr head exists) probably in the mucosa or bony walls the cleft. It is doubtful if the pain can be explai merely on the basis of passive congestion; . (Simpson [63]).

Whaley,[64] however, observed passive congesti immediately after the onset of pain in the e All references to pain by aviators seem not localize it beyond "in the ear." Armstrong stated that the aural pain may radiate to temporal region, the parotid gland and the che In view of the fact that the eustachian tube supplied by the glossopharyngeal nerve, reference of pain in neuralgia of this same ne might be used to infer the location of the " pain" in the "blocked tube." In glossophary geal neuralgia the pain is referred to the skin the area over the mastoid region and the a jacent root of the ear (fig. 30). However, pa originating in the eustachian tube is not report to be felt in this area of skin and so must vaguely localized to "the ear" as though it we in the depths of the middle ear.

Tinnitus occurs in 5 per cent of obstruct tubes [58] and is usually associated with deafne Vertigo occurs slightly less frequently (3 p cent), is likewise associated with deafness a is of peripheral origin.

> Vertigo is caused in most cases, and perhaps in a by unilateral Eustachian obstruction. . . . It see obvious that obstruction of the Eustachian tube sou how disturbs the air pressure and causes stimulation the perilymph, which interferes with normal bala as maintained by labyrinthine mechanism . . . perilymph is disturbed by abnormal variations in pr sure, either increased or decreased.
>
> In many of the cases it has been noted that staggering is in the direction of the obstructed si although this has not been invariably true. . . . any rate the direction of the gait furnishes a clue as the side which may be affected (Merica [64]).

Merica [64] quoted a notation made by Sco in 1936 that when in the examination aviators it was found that both eustachian tub were inefficient and that both tympanic me branes were compressed inward, there was dea ness without vertigo, but that when only o eustachian tube was obstructed vertigo was pro

Ann. Otol., Rhin. & Otolaryng. **51**:995-1006 (Dec.) 1942.

61. Van Dishoeck, H. A. E.: Measurement of the Tension of the Tympanic Membrane and of the Resistance of the Eustachian Tube, Arch. Otolaryng. **34**: 596-602 (Sept.) 1941.

62. Mao, C. Y., and Hughson, W.: Patency of the Eustachian Tube in Relation to Profound Hearing Loss in Childhood, Arch. Otolaryng. **33**:824-829 (May) 1941.

63. Simpson, J. F.: A General Survey of Otorhi logical Considerations in Service Aviation, J. Laryn & Otol. **57**:1-7 (Jan.) 1942.

64. Merica, F. W.: Vertigo Due to Obstruction the Eustachian Tubes, J. A. M. A. **118**:1282-12 (April 11) 1942.

nt, owing to heterogenous stimuli. Richey [65] de similar observations on pilots who after h flying had attacks of vertigo and nausea due unilateral obstruction of the eustachian tube.

(*b*) Signs: Whaley [54] stated:

. The earliest and simplest [sign] is slight reddenof the periphery of the drum and of the handle the malleus. The only distinguishing feature been this aviation condition and early otitis media is fairly pronounced indrawing of the drum. A more anced, or more severe, stage shows diffuse reddenof the drum with fluid bubbles present on the inner face. Later still a fluid level may occur. The fluid transudate, and as such has a black line on top, to old blood cells, and some air is always present.[65a] infection should occur, the transudate becomes an date and the middle ear cavity is completely filled. . . Another picture sometimes presented is that a severely bruised drum and is due to hemorrhage ween the layers of the membrane. An outstanding ture of aero-otitis-media is the suddenness of onset. ave "gone up" in the decompression chamber with advanced stages is directed at the eustachian tube. The nose should be sprayed frequently with a 1 per cent solution of ephedrine, and applicators dipped in the solution may be gently pushed through the nose and allowed to lie against the eustachian orifice and cushion for ten minutes or so. Warm irrigations of the nose and throat with a solution of sodium chloride sometimes lessen the congestion and often add to the comfort of the patient. If, after several days, the condition has not cleared, inflation by means of a eustachian catheter may help; this usually removes any remaining deafness that may result. Should an infection occur and the middle ear fill with pus, paracentesis of the ear drum is necessary.

Farrior [66] has recently substantiated the value of this nontraumatizing, conservative therapy for treatment of disease of the eustachian tube by careful histologic comparisons of normal and recently traumatized tubes (either infected with bacteria or injured mechanically) obtained at autopsy.

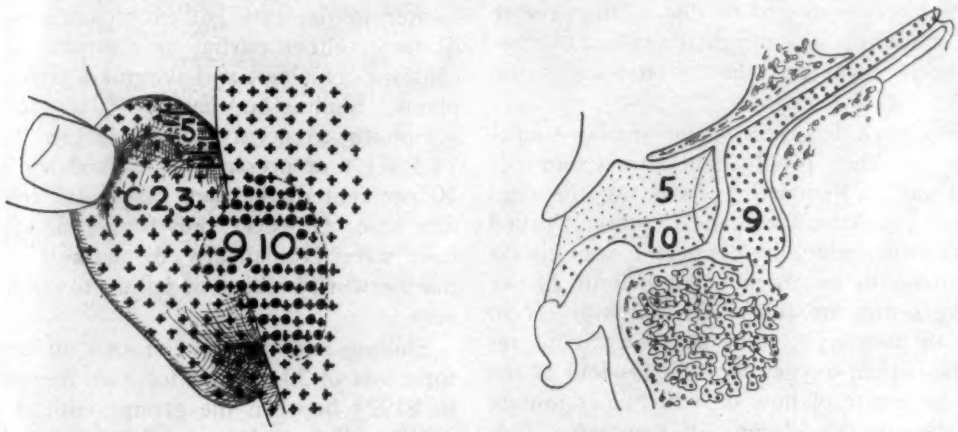


Fig. 30.—A drawing showing the area distribution of the glossopharyngeal nerve (ninth) in the eustachian e, the middle ear, the mastoid cells and the retroaural region of the skin. In the left hand figure, the skin tribution of the fifth nerve (*5*) is indicated by the horizontal lines, the overlapping distribution of the ninth d tenth cranial nerves by the dotted area and the distribution of the second and third cervical nerves (*C 2, 3*) the areas indicated by crosses. In the right hand figure, the areas indicated by the numbers *5*, *9* and *10* present the distributions of the fifth, ninth and tenth cranial nerves, respectively.

oups of airmen to watch the development of this iction, and the moment one complained of pain in the s I examined them and always found the redness. descent from 15,000 feet (4.5 kilometers) to the ound level without the opening of the eustachian tube ll rupture an ear drum. Fortunately, this is an common occurrence, and I know of only 1 case in ich training was stopped for this reason.

Whaley [54] advised:

. . If the inflammation is in the very early stage, is probably best left alone. Any treatment in more

CAISSON WORK

To those who work beneath the surface of the earth the eustachian tube is of as much importance as it is to aviators. The sandhog or caisson worker has more aptly named the disturbance of the tube during compression in the air lock "blocked ear."

Studies of the ear in relation to caisson work are far older than those in relation to aviation. Shilling and Everley [67] pointed out that Heller, Mayer and Von Schrotter in 1900 described temporary deafness and vertigo due to compres-

65. Richey, D. G.: Vertigo, West Virginia M. J. :389-393 (Sept.) 1937.

65a. Since the paper was submitted for publication the otographic appearance of these changes has been blished by Captain I. Hantman: (Secretory Otitis edia [Illustrated by photographs of the tympanic membrane in natural color], Arch. Otolaryng. **38**:561-573 Dec.) 1943.

66. Farrior, J. G.: Histopathologic Considerations in Treatment of the Eustachian Tube, Arch. Otolaryng. **37**:609-621 (May) 1943.

67. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, U. S. Nav. M. Bull. **40**:664-686 (July) 1942.

sion in caisson workers. They likewise recorded a whole series of reports on the effects of compressed air on caisson workers in the years that followed.

Aside from the fact that "blocked ear" is commercially recognized as partially compensable, there is little to distinguish it from the aviation form. Certain discrepancies appear in the descriptions of "blocked ear" as compared with those of the aviation form. Almour [68] could find no characteristic hearing loss in the acute form, the losses ranging from 128 to 8192 double vibrations, although the majority were in the 128 to 512 double vibration range. He observed that the severity of the blockage apparently had nothing to do with the length of time required for the hearing to return to approximately normal—an interval of time ranging from four days to twenty-eight days and averaging sixteen days. This fourfold time of recovery from deafness in caisson workers seems to be due to the greater number of "old hands" and the economic "necessity to keep working," come what may to the ears.

Pioneer work has been done among tunnel workers in the prophylactic treatment of "blocked ear". Requarth,[69] following the suggestions of Lovelace, Mayo and Boothby, worked out a practical technic involving the administration by mask of an 80 per cent helium–20 per cent oxygen mixture at a positive pressure (6 to 32 mm. of mercury). The average patient receives the helium-oxygen mixture for four to ten minutes at a rate of flow of 8 liters per minute and a pressure of 12 mm. of mercury. This treatment is given after the patient leaves the lock as concurrent treatment in the lock is no more effective. The 25 per cent reduction in worker's hours lost and the 75 per cent decrease in number of suppurating ears bear witness to its value. This efficacy of the gas is limited to patients with little or no change in the tympanic membrane and little or no fluid in the middle ear.

SUBMARINE WORK

Not until recently have there been any studies of the effects of increased pressure on the ears of submarine personnel. Prior to entering the water for actual training with the submarine escape appliance, submarine personnel are subjected to 50 pounds' (23 Kg.) air pressure in a recompression chamber. Such a pressure playing on the eustachian orifice is more then three times the atmospheric (2,585 mm. of mercury). In aviation the pressure change is less than doubled.

Shilling and Everley [67] have given the following vivid description of the rapid changes that occur in "blocked" ears in these recompression chambers:

> . . . To the uninitiated, it is truly alarming to look at an ear that a few minutes before was white, glistening and normal and see a fiery red drum so congested and retracted as to cause obliteration of all normal landmarks and covered with bubble-like bulges caused either by air or hemorrhage dissecting the strata of the drum membrane. Often this active congestion and hemorrhage extends onto the walls of the external auditory canal and must also extend into the tympanic cavity for blood at times comes from the eustachian tube in the absence of a rupture of the drum membrane.

Submarine workers [67] with "blocked ears" seem to have symptoms identical with those of caisson workers and aviators—from a feeling of "water in the ears" to excruciating pain, with deafness, either partial or complete, an almost constant complaint and tinnitus a frequent complaint. Shilling and Everley [67] have found the symptoms to occur usually before 10 pounds (4.5 Kg.) of pressure is reached and in about 10 per cent of the men taking the test for the first time. However, only 9 of 4,333 officers and men were permanently disqualified from submarine work because of failure to equalize pressure.

Shilling and Everley [67] found an almost uniform loss of 20 decibels for each frequency (128 to 8192) between the groups with acute aero-otitis media and the normal groups of submarine personnel. In the cases of aero-otitis media the hearing usually returned to normal within a few days, although in some the recovery was delayed for months, and a few never showed a complete return to normal hearing.

Deep sea divers in the naval service rarely have "blocked ear," for they are dropped slowly to the depths and their exposure to high pressures is gradually incurred. In Shilling and Everley's [67] experience aero-otitis media and the associated loss of hearing do not develop in deep sea divers.

TREATMENT OF INEFFICIENCY OF THE EUSTACHIAN TUBE

Various investigators of blocking of the eustachian tube have sought to bring about normalcy in numerous ways. Direct treatment has been used in the form of politzeration,[70] inflation

68. Almour, R.: The "Blocked Ear" of the Caisson Worker, Laryngoscope **52**:75-81 (Jan.) 1942.

69. Requarth, W. H.: Aero-Otitic Media in Compressed Air Workers: Treatment with Helium-Oxygen Mixtures, J. A. M. A. **116**:1766-1769 (April 19) 1941.

70. Politzer, A.: Ueber ein neues Heilverfahren gegen Schwerhörigkeit in Folge von Unwegsamkeit der Eustachischen Ohrtrompete, Wien. med. Wchnschr. **13**:[illegible] 102, 117 and 148, 1863.

in by means of a catheter,[71] the Valsalva ethod,[72] use of a heat bougie,[73] irradiation,[74] flation with benzedrine vapor[75] and irrigan of the tympanic cavity,[76] even massage.[77] owever, conservative treatment seems to be e method of choice after enthusiasms have ned. Anthony[78] found that inflation of the tubes was contraindicated because of the frequently associated inflammation of the eustachian tubes. Shilling and Everley[67] maintained that warm, soothing oil in the external ear gave as much relief as attempts to equalize air pressure by means of the eustachian catheter or the politzer bag. Shrinking of the eustachian orifice by application of an aqueous solution of an ephedrine salt or a similar vascular constrictor in addition to the factor of passing time seems to yield the best results. The value of nontraumatizing, conservative treatment is substantiated by the histologic studies of Farrior.[66]

For the opposite condition, in which the eustachian tube is continuously open, Shambaugh[22] mentioned Bezold's method of closing the tube for one to two weeks by the irritant action of a little powder composed of 1 part salicylic acid and 4 parts of boric acid insufflated into the tube through a eustachian catheter. Some, according to Shambaugh, are able to dispense with the salicylic acid powder by the simple snuffing of isotonic solution of sodium chloride through the nose once or twice a day to stimulate the atrophic mucosa. Head colds give immediate and lasting relief for the duration.

71. Guyot, 1724, cited by Garrison, F. H.: History Medicine, Philadelphia, W. B. Saunders Company, 29, p. 352. Cleland A.: Instruments Proposed to emedy Some Kinds of Deafness Proceeding from struction in the External and Internal Auditory ssages, Phil. Tr., London, 1732-1744, p. 124; cited Garrison.

72. Valsalva, A. M.: De aure humana tractatus, aj. ad Rhenum, G. vande Water, 1717.

73. Simon, W. E.: Treatment of Chronic Catarrhal afness with the Eustachian Heat Bougie, Arch. olaryng. **27**:606-617 (May) 1938.

74. Schenck, H. P.: The Influence of Nasopharyngeal yperplasia on the Ear: Histological Examination of yperplastic Lymph Follicles After Irradiation, Tr. m. Otol. Soc. **31**:48-61, 1941.

75. Sulman, L. D.: Treatment of Blockage of the stachian Tube by Inflation with Amphetamine Vapor, rch. Otolaryng. **35**:646-649 (April) 1942.

76. Leasure, J. K.: Tympanic Cavity Irrigation, Ann. ol., Rhin. & Laryng. **51**:253-256 (March) 1942.

77. Lewy, A.: Massage of the Eustachian Tube, Ann. ol., Rhin. & Laryng. **25**:898-903 (Dec.) 1916.

78. Anthony, D. H.: A Review of the Literature on juries to Eyes, Ears and Sinuses and a Summary of Seventy Cases Treated, Memphis M. J. **14**:76-79 (May) 1939.

BACILLUS PROTEUS INFECTIONS

REVIEW OF THE LITERATURE AND REPORT OF A CASE OF SEPTICEMIA OF OTITIC ORIGIN TREATED WITH SULFAPYRIDINE, WITH RECOVERY

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Reports of infections due to Bacillus proteus have appeared in the literature occasionally for many years. Until 1920 there was considerable controversy as to whether this organism was ever pathogenic. Most observers now feel that it is often a saprophyte but that when pathogenic it is extremely toxic.

CHARACTERISTICS OF THE ORGANISM

B. proteus is a member of the colon group of bacilli and hence is usually present in feces and in sewage. Proteus vulgaris and Proteus mirabilis are the most important members of the Proteus group from a clinical standpoint.

According to Zinsser, the proteus bacillus is gram-negative, motile and non-spore-bearing. It liquefies gelatin and ferments dextrose and saccharose with gas. On gelatin plates it shows colonies characterized by spreading streamers. Occasionally it has been found to be the causative organism of dysentery, infections of the urinary tract and war wounds. Less frequently it has caused otitis media, septicemia, meningitis and abscess of the brain.

REVIEW OF THE LITERATURE

The proteus bacillus was first described by Hauser in 1885. He found it in decomposed meat and animal matter. Walter Reed in 1894 reported one of the earliest cases in which the organism was thought to be pathogenic, a case of pneumonia in which both the proteus bacillus and Diplococcus lanceolatus were found in the sputum. At autopsy large areas of the lung showed a pure culture of proteus bacillus.

Ware in 1902 reported a case of abscess of the mouth in which the patient recovered after incision and drainage.

INFECTIONS OF THE URINARY TRACT

The urinary tract is the most common location for B. proteus infections. No attempt was made to collect all cases of infection of the urinary tract, because they were too numerous. Kretschmer and Mason, in a series of 305 cases of such infection, found B. proteus present in 28. It was associated with other organisms in cases and was found in pure culture in 8.

Hager and Magath, Walters and Scott, a Kapusto have discussed various phases of infe tion of the urinary tract.

MacKenzie and Hawthorne reviewed the lite ature and reported a case of pyonephrosis. Th found 17 cases of septicemia and reported 1 their own. In 5 of the cases the patients di These authors' paper is one of the most comple on this type of B. proteus infection.

The prognosis is good in cases of uncomp cated infections of the urinary tract. In cas in which there are complications it is much bett than in cases in which the primary infection in some other site.

WAR WOUNDS

The proteus bacillus was found fairly fr quently in war wounds. Goadby found it 8 to 20 per cent of cases of such wounds. T odor of the discharge was foul. The prote bacillus was associated with other organisms most cases and was probably a saprophyte in so instances.

OSTEOMYELITIS OF VERTEBRAE

In 1934 Selig reported the first case of oste myelitis of vertebrae due to the proteus bacill No cases have been described in the literatu since that time. In his case the condition fo lowed a cystoscopic examination and was hematogenous origin. The bodies of two c vical vertebrae were involved. After eig months recovery occurred, with solid fusion the vertebrae.

OTITIS MEDIA

In most of the cases of otitis media in wh B. proteus has been found the condition h been chronic, of several years' duration.

Some observers feel that other organis caused the otitis media at the onset. This not definitely established, however. That t proteus bacillus was the pathogenic organi in the reported cases there can be little or doubt, for it was found in pure culture in ma of them.

The clinical course was characterized by rapid d extensive destruction of the mastoid. The eral sinus and the dura were exposed by the structive process in many cases, even though nical symptoms had been present only for a days. Thrombosis of the lateral sinus with sitive blood culture was found in a large pro-

TABLE 1.—*Otitis Media*

Name of Observer	Date Reported	Septicemia	Meningitis	Abscess of Brain	End Result
lmacher	1897	No	No	Yes	Died
es	1912	No	Yes	No	Died
uer	1918	No	Yes	No	Died
nelli	1920	Yes	No	No	Died
rnen	1922	Yes	No	No	Died
monoui et al	1925	Yes	No	No	Died
llagher	1929	No	No	Yes	Recovered
rtenhaus	1930	Yes	Yes	No	Died
in	1930	No	No	No	Recovered
gniez et al	1932	Yes	Yes	No	Died
ith et al	1935	Yes	No	No	Died
ngalos et al	1935	?	Yes	No	Died
lhoun	1936	No	Yes	No	Recovered
Govern	1936	Yes	No	No	Died
Govern	1936	Yes	No	No	Died
llée et al	1937	Yes	No	No	Died
uquin	1939	No	Yes	No	Recovered
rzog	1939	Yes	No	No	Recovered
rmin et al	1940	Yes	No	Yes	Recovered
ter et al	1940	Yes	Yes	No	Died
gg	1941	Yes	Yes	No	Recovered

rtion of cases. Meningitis and abscess of the in were much less frequent complications were fairly often present. Table 1 lists all the cases that could be found in the literature d shows the complications.

BLE 2.—*Cases of Aural Disease in Which the Patient Recovered*

Reported by	Date Reported	Septicemia	Meningitis	Abscess of Brain	Therapy
llagher	1929	No	No	Yes	Surgical
n	1930	No	No	No	Surgical
lhoun	1936	No	Yes	No	Surgical
rzog	1939	Yes	No	No	Surgical and stock bacteriophage
rmin et al	1940	Yes	No	Yes	Surgical
uquin	1940	No	Yes	No	Surgical and sulfapyridine
gg	1941	Yes	Yes	No	Surgical, sulfanilamide and sulfapyridine

The patients survived in only 7 of the 21 orted cases. Surgical intervention and the al postoperative management were used in cases. In addition, Gerzog used stock bacophage and felt it was effective. Mouquin ployed sulfapyridine with striking improvent. Neter and Chait used sulfanilamide in dition to surgical treatment, in their case without benefit. Cragg used sulfapyridine first, without benefit, and later sulfanilamide, with slight to moderate benefit.

MENINGITIS

Eighteen cases of meningitis due to Bacillus proteus have been described in the literature. The sites of the lesion that caused the meningitis varied in different cases; however, otitis media was much the most common cause.

TABLE 3.—*Meningitis*

Name of Observer	Date Reported	Cause	Complications	Result
Ross	1912	Otitis media	.....	Died
Goebel	1914	Not determined	.....	Died
Bauer	1918	Otitis media	.....	Died
Anderson	1921	Not determined	.....	Died
Anderson	1921	Not determined	.....	Recovered
Kernen	1922	Peritonsillar abscess	Brain abscess	Died
Bischoff et al.	1925	Not determined	.....	Died
Bewley et al.	1927	Not determined	.....	Died
Neal et al.	1927	Not determined	.....	Died
Neal	1927	Pneumonia	.....	Died
Kortenhaus	1930	Otitis media	Septicemia	Died
Pagniez	1932	Otitis media	Septicemia	Died
Cathala et al.	1933	Not determined	.....	Died
Pangalos et al.	1935	Otitis media	.....	Died
Calhoun	1936	Otitis media	.....	Recovered
Mouquin	1939	Otitis media	.....	Recovered
Neter et al.	1940	Otitis media	Septicemia	Died
Cragg	1941	Otitis media	Septicemia and lung abscess	Recovered

The onset of the meningitis was sudden and the disease terminated fatally in a few days in most cases. Table 3 gives data from all such cases of which reports could be found in the literature.

The treatment was directed toward the lesion that caused the meningitis, if it could be determined. In 4 of the 18 cases the patients recovered.

ABSCESS OF THE BRAIN

Four cases of abscess of the brain due to Bacillus proteus have been reported (table 4).

TABLE 4.—*Abscess of the Brain*

Name of Observer	Date Reported	Cause	Complications	Result
Ohlmacher	1897	Otitis media	.....	Died
Kernen	1922	Peritonsillar abscess	Meningitis and septicemia	Died
Gallagher	1929	Otitis media	.....	Recovered
Dearmin et al.	1940	Otitis media	Septicemia	Recovered

The abscess followed otitis media in 3 cases and peritonsillar abscess in 1. The abscesses of the brain were drained surgically.

SEPTICEMIA DUE TO CAUSES OTHER THAN INFECTION OF URINARY TRACT

There have been 23 reported cases of septicemia due to Bacillus proteus from causes other

than infection of the urinary tract. Otitis media was the most common cause.

The course of the disease has been short and the termination fatal in most cases. The treatment has been directed toward the disease causing the septicemia, if that could be determined. Sulfanilamide was used in 1 case by Neter and Chait, but the condition terminated fatally. Craig used sulfapyridine first without benefit and later sulfanilamide with slight to moderate benefit.

E. G., a 53 year old man, complaining of earache and malaise, was seen on July 4, 1939. For two days he had been having pain in muscles and joints and had been in bed. The right ear drum was red and bulging. The temperature was 102 F., and he appeared to be acutely ill. The results of examination were normal in other respects. He was taken to St. Joseph's Hospital, Keokuk, Iowa, for treatment.

TABLE 5.—*Septicemia from Causes Other than Infection of Urinary Tract*

Name of Observer	Date Reported	Cause	Complications	End Result
Stinelli.....	1920	Otitis media	.....	Died
Kernen.....	1922	Peritonsillar abscess	Brain abscess and meningitis	Died
Kernen.....	1922	Otitis media	.....	Died
Warren et al...	1924	Mouth infection	.....	Died
Irimonoui et al	1925	Otitis media	.....	Died
Assis de.....	1927	Not reported	.....	Recovered
Herzig.....	1928	Not determined	.....	Died
King et al.....	1929	Lung abscess	.....	Died
Stein.....	1930	Following radium applications to tonsils	.....	Died
Kortenhaus....	1930	Otitis media	.....	Died
Pagniez et al...	1932	Otitis media	Meningitis	Died
Paryzek et al..	1932	Induced labor and pyelitis	.....	Recovered
Smith et al.....	1935	Otitis media	.....	Died
Decourt et al...	1936	Not determined	.....	Died
McGovern.....	1936	Otitis media	.....	Died
McGovern.....	1936	Otitis media	.....	Died
Vallée et al.....	1937	Otitis media	.....	Died
Livramento....	1938	Not determined	.....	Died
Baudet.....	1939	World War wound	.....	Died
Gerzog.....	1939	Otitis media	.....	Recovered
Neter et al.....	1940	Otitis media	Meningitis (sulfanilamide used)	Died
Dearmin et al..	1940	Otitis media	Brain abscess	Recovered
Cragg.....	1940	Otitis media	Meningitis and lung abscess	Recovered

The family history was irrelevant.

History.—In 1937 he had acute mastoiditis with sinus thrombosis on the right side. I did a simple mastoidectomy and opened the lateral sinus. There was free bleeding from above when the sinus was opened, but none from below. The clot was removed from below with a suction tip, and then the sinus bled freely. The patient recovered after a stormy postoperative course. Cultures of blood and of material from the mastoid were negative. He later had several attacks of acute otitis media, but each time he recovered without complications. Hearing was essentially normal in April 1939.

Operation and Course of Illness.—A paracentesis of the right ear drum was done. There was profuse purulent discharge, which was light brown. Bacteriologic examination of the discharge revealed a pure culture of a bacillus of the colon group, which was later identified as B. proteus. On July 5 a culture of blo was made and sulfanilamide in a dose of 20 gra (1.3 Gm.) every four hours was given by mouth. So cyanosis developed, but no other change in the cou of the disease was observed. The blood culture show B. proteus. Culture was repeated twice at daily interv and each time showed a pure growth of B. prote The temperature now varied from 102 to 103 F., and t patient was steadily losing ground. Two days la the sulfanilamide therapy was discontinued and adm istration of sulfapyridine in the same dosage w started. The temperature returned to normal in l than twenty-four hours and was never more th slightly elevated afterward. There was a moder amount of nausea and vomiting, but there were other toxic symptoms due to the drug.

The dosage of the drug was gradually reduced, a its use was finally discontinued after two weeks. T ear stopped draining a few days after treatment w sulfapyridine was started.

Since that time the patient has had recurrent atta of otitis media, but after each one resolution w complete.

In February 1940 some swelling and fluctuation dev oped in the region of the old scar after an attack of ot media. A second operation on the mastoid was p formed, but little except granulation tissue was fou

In June 1941, when I entered the military servi the patient was in fair condition. He became tir rather easily and was unable to work as much as would like. He had no particular symptoms refera to the ear.

There have been 3 such cases in which t patients were treated with sulfanilamide a allied compounds. Neter and Chait treated patient with mastoiditis complicated by sep cemia and meningitis with sulfanilamide, but t condition terminated fatally. In a case of oti meningitis Mouquin used sulfapyridine, and t patient recovered. Cragg reported a case which mastoiditis was complicated by meningi septicemia and abscess of the lung. After oper tion sulfapyridine was used for five days, wi out clinical improvement. Later sulfanilami was used, and clinical improvement was gradu It was felt that the sulfanilamide therapy w helpful, but the response to the drug was n striking.

Sulfanilamide was ineffective in the case have reported here, but improvement was rap after sulfapyridine was given. Carey stated th sulfanilamide and sulfathiazole are the only s fonamide compounds effective in the treatme of B. proteus infections of the urinary tract.

SUMMARY

A review of the available literature on Bacil proteus infections of all sites except the urina tract revealed that meningitis, septicemia a abscess of the brain are the most serious ma festations of the disease. They are most oft due to otitis media and mastoiditis.

The urinary tract is the most common site for proteus infections. The prognosis is good these cases.

The mortality rate is high with infections of sites except the urinary tract.

Sulfapyridine is apparently much more effec- e than sulfanilamide in treatment of B. proteus fections.

BIBLIOGRAPHY

derson, J. M.: Proteus in Cerebro-Spinal Fluid of Typhus, J. Path. & Bact. **24**:478 (Oct.) 1921.

Assis, A.: Proteus Pseudovaleriae and Its Occurrence in Man, J. Hyg. **27**:108 (Nov.) 1927; Bol. Inst. Vital Brasil **1**:3, 1927.

uer, A.: Acute Purulent Meningitis Caused by a Proteus (Bacillus Submobilis), Compt. rend. Soc. de biol. **81**:745, 1918.

udet, G.: Septicemia Due to Bacillus Proteus: Case, Mém. Acad. de chir. **65**:921 (June 21) 1939.

wley, G., and Horgan, E. S.: Case of Meningitis Associated with B. Proteus, Lancet **1**:819 (April 16) 1927.

schoff, H., and Brekenfeld: Proteus Meningitis in Infant, Ztschr. f. Kinderh. **39**:421, 1925.

ckley, T. I.: Bacillus Proteus Septicemia, California & West. Med. **38**:436 (June) 1933.

lhoun, J. C.: Meningitis Due to Bacillus Proteus: Case with Recovery, Canad. M. A. J. **34**:679 (June) 1936.

rey, B. W.: Use of Sulfanilamide and Related Compounds in Diseases of Infancy and Childhood, J. A. M. A. **115**:924 (Sept. 14) 1940.

thala, J., and Gabriel, P.: Meningitis in New-Born Due to Infection with Proteus Vulgaris, Bull. Soc. de pédiat. de Paris **31**:174 (April) 1933.

oper, F. B.; Gross, P., and Lewis, M.: Sulfonamide (Sulfanilamide and Sulfapyridine) Therapy of Experimental Peritonitis Due to Escherichia Coli: Bacillus Proteus and Bacillus Pyocyaneus, Proc. Soc. Exper. Biol. & Med. **40**:34 (Jan.) 1939.

gg, N. R.: Otitic Meningitis Due to Bacillus Proteus, Arch. Otolaryng. **34**:345 (Aug.) 1941.

armin, R. M., and Sims, J. L.: Thrombosis of Lateral Sinus and Abscess of the Temporal Lobe Due to Bacillus Proteus, Arch. Otolaryng. **32**: 1098 (Dec.) 1940.

court, J.; Lévy-Bruhl, and Franchel: Pseudomalarial Form of Septicemia Due to Bacillus Proteus; Recovery of Case, Bull. et mém. Soc. méd. d. hôp. de Paris **52**:65 (Jan. 27) 1936.

llagher, J. R.: Bacillus Proteus Brain Abscess, Yale J. Biol. & Med. **2**:143 (Dec.) 1929.

rzog, B. G.: Bacillus Proteus in Otogenic Infections, Secondary Mastoiditis, Thrombosis of Lateral Sinus and Bacteremia, Arch. Otolaryng. **30**: 275 (Aug.) 1939.

adby, K.: Septic Wounds, Lancet **2**:89 (July 15); 585 (Sept. 30); 851 (Nov. 18) 1916.

bel, F.: Proteus Meningitis and Proteus Sepsis in a New-Born with Remarks on the Proteus as a Causative Agent of Disease in Man, Deutsches Arch. f. klin. Med. **116**:119, 1914.

ger, B. H., and Magath, T. B.: Four Cases of Urinary Calculi from Which Proteus Ammoniae Was Isolated, M. Clin. North America **10**:693 (Nov.) 1926.

Hauser, G.: Bacteria, Proteus Group: Sepsis Complicated with Appendicitis; Case, Deutsche Ztschr. f. d. ges. gerichtl. Med. **21**:300, 1933.

Herter, C. A.: A Biochemical Study of Proteus Vulgaris Hauser, J. Biol. Chem. **9**:491, 1911.

Herzig, H.: Proteus Meningitis in Infant; Case, Ztschr. f. Kinderh. **45**:443, 1928.

Irimonoui, G., and Popa, R.: Proteus Vulgaris Causing Septicemia in Man, Compt. rend. Soc. de biol. **82**: 1082 (April 10) 1925.

Kernen, J. D., Jr.: Two Cases of Infection by Bacillus Proteus, Laryngoscope **32**:304 (April) 1922.

King, M. J., and Morgan, R. H.: Multiple Pulmonary Abscesses Resulting from Blood Infection by Microorganisms of Proteus Group, Am. Rev. Tuberc. **19**: 182 (Feb.) 1929.

Kortenhaus, F.: Otogenic Meningitis Due to Bacillus Proteus: Case, Ztschr. f. Laryng., Rhin. **19**:146 (Jan.) 1930.

Kretschmer, H. L., and Mason, L. W.: Bacillus Proteus Septicemia: Two Cases with Recovery, J. A. M. A. **92**:1734 (May 25) 1939.

Larson, W. P., and Bell, E. T.: A Study of the Lesions Produced by Bacillus Proteus, J. Infect. Dis. **13**: 510, 1913.

Livramento, F.: Proteus Mirabilis Isolated from Blood: Case, Hospital, Rio de Janeiro **14**:1393 (Dec.) 1938.

MacKenzie, D. W.: Bacillus Proteus Pyonephrosis with Blood Infection: Review of Literature and Report of Case with Operation and Recovery, Tr. Am. A. Genito-Urin. Surgeons **26**:333, 1933.

——and Hawthorne, A. B.: Pyonephrosis with Blood Infection: Review of Literature and Report of Case with Operation and Recovery, J. Urol. **30**:277 (Sept.) 1933.

McGovern, F. H.: Bacteria, Proteus Group: Fatal Otogenic Infection; Two Cases, Arch. Otolaryng. **24**:618 (Nov.) 1936.

Meltzer, T.: Bacillus Proteus Meningoencephalitis, Internat. Clin. **50**:96 (March) 1941.

Mouquin, M.; Plichet, A., and Beuvier, J.: Acute Otogenous Meningitis Due to Proteus Vulgaris Cured by Sulfapyridine, Bull. et mém. Soc. méd. d. hôp. de Paris **55**:1271 (Nov. 10) 1939.

Neal, J. B., and Abramson, H.: Bacteria, Proteus Group, in Meningitis, Am. J. M. Sc. **174**:665 (Nov.) 1927.

Neter, E., and Chait, R. A.: Bacillus Proteus in Otitis Media, Complicated by Mastoiditis, Mennigitis and Bacteremia, Arch. Otolaryng. **32**:947 (Nov.) 1940.

Ohlmacher, A. P.: The Bacterium Vulgare in a Case of Cerebellar Abscess and Leptomeningitis Following Middle Ear Diseases, Cincinnati Lancet & Clinic **39**:221, 1897.

Pagniez, P.; Plichet, A., and Decourt, P.: Fatal Septicemia Caused by Proteus Vulgaris: Case, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:270 (March 7) 1932.

Pangalos, G. E., and Doucas, J.: Fatal Primary Meningitis Caused by Proteus Vulgaris, Bull. et mém. Soc. méd. d. hôp. de Paris **51**:1436 (Nov. 4) 1935.

Paryzek, H. V., and Ecker, E. E.: Bacteria, Proteus Group: Septicemia with Recovery, Am. J. M. Sc. **184**:533 (Oct.) 1932.

Pinichet, G. C.; Castillo, B. D., and Cruz, J. A. M.: Septicemia Due to Bacillus Proteus in an Infant Eight Months Old: Case and Recovery, Bol. Soc. cubana de pediat. **11**:718 (Dec.) 1939.

Reed, W.: Association of Proteus Vulgaris with Diplococcus Lanceolatus in a Case of Croupous Pneumonia, Bull. Johns Hopkins Hosp. **5**:24, 1894.

Ross, E. A.: A Case of Meningitis in Which the Only Organism That Could Be Cultivated from Cerebro-Spinal Fluid During Life Was a Bacillus of Proteus Group, Lancet **2**:1292, 1912.

Selig, S.: Bacillus Proteus Osteomyelitis of Spine, J. Bone & Joint Surg. **16**:189 (Jan.) 1934.

Smith, A. B. P., and Miller, S.: Lateral Sinus Thrombosis Due to Bacillus Proteus, Lancet **1**:862 (April 13) 1935.

Stein, S.: Bacillus Proteus Infection in Secondary Mastoiditis, Following Tonsillectomy, and in Radium Implantation in Tonsils, Ann. Otol., Rhin. Laryng. **39**:1155 (Dec.) 1930.

Stinelli, F.: Primary Suppurative Mastoiditis Caus by Proteus Vulgaris, Gazz. internaz. med.-chir. 2 197, 1920.

Vallée, A., and Giroux, M.: Acute Mastoiditis D to Proteus Vulgaris, Complicated by Septicem and Purulent Pleurisy: Case, Laval méd. **2**:1 (April) 1937.

Walters, W., and Scott, D. E.: Case of Prote Ammoniae Septicemia with Recovery, Proc. St Meet., Mayo Clin. **5**:168 (June 18) 1930.

Ware, M.: The Role of the Bacillus Proteus Vulgar in Surgery, Ann. Surg. **36**:120, 1902.

Warren, S., and Lamb, M. E.: Fatal Infection wi an Organism of Proteus Group, J. M. Researc **44**:375 (March) 1924.

Zinsser, H.: A Text Book of Bacteriology, ed. 6, Ne York, D. Appleton and Company, 1931, p. 543.

CONGENITAL LARYNGEAL STRIDOR (INSPIRATORY LARYNGEAL COLLAPSE)

A NEW THEORY AS TO ITS UNDERLYING CAUSE AND THE DESIRABILITY OF A CHANGE IN TERMINOLOGY

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Present day interest in the problem of stridorous breathing in newborn and older infants received its major impetus from Lees's report on a Larynx from an Infant with a Peculiar Form of Obstructed Respiration." This report was given the London Pathological Society in 1883.

Although it is a well established fact that Lees was not the first to describe the typical condition which one associates with congenital laryngeal stridor, he apparently was the first to show the defective laryngeal mechanism which produces the stridor. The illustration contained in the published report is remarkable for its accuracy and clarity (fig. 1). Lees posed the problem of the underlying causation of the condition—a problem which remains unsolved today, even though the literature abounds with theories and writings relating thereto. It is to Lees's credit that he clearly stated the signs and symptoms of the condition, and it is noteworthy that the ill chosen name "congenital laryngeal stridor" was not proposed by him nor was it used in connection with the condition for years after his report. Sutherland and Lack[2] in their familiar publication on the subject in 1897 used the title "Congenital Laryngeal Obstruction."

Thomson and Turner[3] in their report, which was largely a critique of the previous publications of Lees and of Sutherland and Lack, referred to the condition as "congenital stridor of infants." Subsequent writers on the subject consolidated the wording to "congenital laryngeal stridor," which has survived to this day.

Thomson and Turner conducted experiments on larynxes from infants by placing open tubing from below upward through the glottis and applying suction, thereby attempting to reproduce artificially the phenomena which were observed laryngoscopically in such cases. They concluded their publication with an opinion which was destined to have great influence on later thought and study on this subject:

> . . . It may be well that we should add a few words to define as clearly as possible how our position differs from that held by Drs. Sutherland and Lack in their paper already referred to, which seems to us by far the most important contribution made on the subject. We are cordially at one with them when they say that the affection depends on a valvular action of the upper aperture of the larynx, a falling in, due to "the flaccidity of the parts of the infants." We join issue with them altogether, however, when they ascribe primary etiological importance to the peculiar conformation of the upper aperture found in these cases and regard it as a congenital malformation. We consider it an acquired deformity and hold that it, as well as the stridor, are the result of the ill-coordinated stammering breathing.

This placed the condition on a neurologic basis, whereas Sutherland and Lack properly concluded that it was purely an anatomic anomaly.

A review of subsequent medical literature shows that as a result of this contention the pendulum of opinion and investigation was swung away from the conclusion of Sutherland and Lack. A great many theories have been advanced to explain the observation on a basis compatible with the dissenting opinion of Thomson and Turner. As a consequence, various disorders of the respiratory apparatus have been advanced as "causes" of "congenital laryngeal stridor." This accumulation of causes today embraces dysfunctions of neuropathic, myopathic, nutritional, inflammatory, endocrine, neoplastic or other origin, stridor alone apparently being the sine qua non for inclusion. "Congenital laryngeal stridor" as the term is now used no longer connotes a single physical or clinical entity.

The pendulum of opinion is returning to the original conception of Sutherland and Lack. Since two schools of thought on the matter exist it behooves one to state for the sake of clarity when referring to "congenital laryngeal stridor" whether one is referring to the prototype as implied by Lees and by Sutherland and Lack

1. Lees, D. B.: Larynx from an Infant with a Peculiar Form of Obstructed Respiration, Tr. Path. Soc. London **34**:19, 1883.
2. Sutherland, G. A., and Lack, H. M.: Congenital Laryngeal Obstruction, Lancet **2**:653, 1897.
3. Thomson, J., and Turner, A. L.: On the Causation of the Congenital Stridor of Infants, Brit. M. J. 1:561, 1900.

or to a symptomatologic diagnosis embracing many varieties of stridorous breathing encountered in infants of which the prototype is but one, if the most frequent, variety.

In order to clarify the extremely confused situation that exists today it would be highly desirable that the term "congenital laryngeal stridor" be done away with and one more distinguishing and explicit given to the condition which is under consideration. The term is, moreover, a throwback to early medical times when diagnosis was necessarily inferential owing to lack of direct methods of inspection and diagnosis which are available today. Direct laryngoscopy as now practiced provides an exact means. Nevertheless, the error persists. It would be no more improper from a nomenclatural standpoint

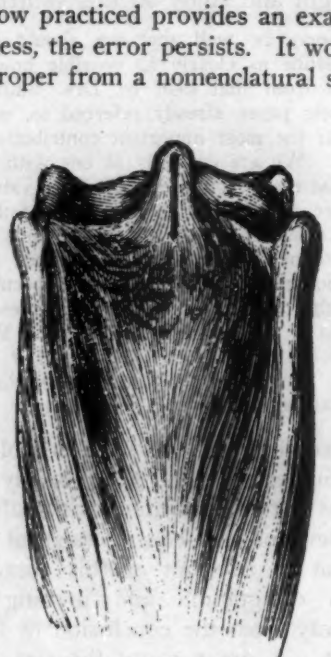

Fig. 1.—Illustration (woodcut) from Lees's original report showing the abnormal character of the larynx in an infant with what he termed a "peculiar form of obstructed respiration." It was many years afterward that the term "congenital laryngeal stridor" came into use. Note the slitlike laryngeal aperture.

to accept as correct such a diagnostic name as "acquired pulmonary wheezing" and thereafter to list as causes bronchial asthma, endobronchial foreign body, benign and malignant neoplasms and all other morbid states in which wheezing is present. Instead, and properly, physicians recognize wheezing as an objective finding common to the various clinical entities mentioned. So it should be with stridor, which should be reckoned as purely an objective sign of numerous and often unrelated conditions.

Among the conditions which have been listed as "causes" of stridor are: paralysis or paralyses of the vocal cords, webs, tumors, hypertrophi or swellings of one or more portions of the en larynx, dislocations or fixations of the cricoar tenoid joints, pseudosyrinx, congenital goiter cretinism, laryngomalacia and flaccid epiglot Each of these is itself an entity and in each t stridor is produced in a different mechanical anatomic manner. For example: a tumor, hyp trophy or swelling causes stridor by reducing t caliber of the respiratory lumen, and strid might be observed in both phases of respirati In palsy of the vocal cords and in fixation of t cricoarytenoid joints the stridor is due to t failure of the abductor mechanism to functi In the condition under consideration, as will seen, the stridor is produced in still anoth manner and is present only on inspirati These few examples are cited to illustrate t impossibility of explaining so protean a con tion as "congenital laryngeal stridor" on t basis of a single mechanism as suggested Thomson and Turner.

INSPIRATORY LARYNGEAL COLLAPSE

Since no name exists which identifies the pro type exclusively, "inspiratory laryngeal collaps is suggested and will be used herein for th purpose. Heatly [4] stated that the use of "co genital laryngeal stridor" should be reserved f this entity and that all other types of stridoro breathing should be excluded from this classi cation. Jackson [5] identified the entity as "lary gomalacia." Kernan [6] in his reecnt work w content to state that it is the condition mo typical of "congenital laryngeal stridor." The varied points of view bespeak the existing co fusion and emphasize the need for reconsider tion of the subject. Either the suggestion Heatly should be taken or, better still, a ne and more exactly descriptive term should adopted. The one herein used suggests itself me as adequate for this purpose.

Most important is the need of determini what is the primary etiologic factor underlyi the laryngeal collapse. This is the poignant que tion raised by Lees and Sutherland and by La from the outset. It is on this point alone that th problem remains unsolved to this day. Invest gators know what the mechanism is but they se the underlying factor which gives rise to th mechanism.

4. Heatly, C. A.: Larynx in Infancy: Study Chronic Stridor, Arch. Otolaryng. **29**:90 (Jan.) 19

5. Jackson, C., and Jackson, C. L.: Diseases a Injuries of the Larynx, New York, The Macmill Company, 1942.

6. Kernan, J.: Loose-Leaf Medicine of the No and Throat, New York, Thos. Nelson & Sons, 19

The title selected, "Inspiratory Laryngeal Collapse," is graphic and descriptive of what is observed by direct laryngoscopy. The mechanism was well described and illustrated many years ago (fig. 2). The earlier observers were limited to mirror images and therefore were not able to exclude an endolaryngeal pathologic condition as a coexisting factor to be reckoned with. A recent experience with a case of inspiratory laryngeal collapse (figs. 3 and 4) afforded me the opportunity of having colored motion pictures taken of this condition through direct laryngoscopy. So far as I know, these are the first motion pictures taken of this anomaly. Characteristic views of each phase of inspiration are herewith presented and provide a pictorial basis for a description of the malady (fig. 5).

Fig. 2.—Larynx from a 2½ month old infant with "congenital laryngeal stridor." The infant died of pneumonia. This case was reported by H. Refslund (Ueber Respirationsstörungen in Folge von Missbildung der Epiglottis, München. med. Wchnschr. **43**:1182, 1896).

CLINICAL MANIFESTATIONS AND CHARACTERISTICS

It must be borne in mind that there are varying degrees of this condition and that the objective findings, while consistent, are proportionate in severity to the degree of laryngeal occlusion. In some cases the condition is so mild as to pass unnoticed until even slight edema of the parts from an intercurrent infection of the upper respiratory tract renders it no longer asymptomatic. Profound collapse is noticeable from birth.

Stridor.—Stridor is always present and the intensity, pitch and character vary with the amount of obstruction. It has been variously described as simulating purring, croaking or some other type of sound. The variety and the volume of sound produced is of little importance except to denote the degree of obstruction, in a given case. The stridor is produced by the flapping together of the epiglottidean edges, the aryepiglottic folds and the soft tissues surmounting the arytenoid eminences. It differs little, if at all, from that observed in other forms of obstructive laryngeal dyspnea as regards distinguishing characteristics of tones or other qualities. It is accentuated by crying, struggling, sudden exposure to cold or other measures that increase the respiratory intake.

Fig. 3 (case 3).—Five month old infant with characteristic "congenital laryngeal stridor" demonstrating obstructive laryngeal dyspnea. On inspiration stridor was marked. Note the indrawing of the xiphoid process. Cyanosis was absent, and this differentiated the condition from other forms of obstructive laryngeal dyspnea. Note the receding chin.

Fig. 4 (case 3).—A close-up view emphasizing the receding or micrognathic jaw.

Respiration.—Inspiration is obstructed. Expiration is unobstructed. Stridor is consequently heard only on inspiration. This is extremely important, for it differentiates the condition from numerous others in which stridor is present on

expiration or during both phases of respiration. The classic signs of obstructive laryngeal dyspnea are evident; these are indrawings of the xiphoid process, intercostal spaces and suprasternal notch. The amount of indrawing, like the stridor, is commensurate with the obstruction. The respiratory rate is increased as a result of the diminished intake of air per inspiration. This is a compensatory adjustment. Cyanosis, as a rule, is absent; this is important in distinguishing the condition from sudden and alarming obstructive laryngeal dyspnea of inflammatory or foreign body origin which calls for immediate tracheotomy or other adequate measures of relief if life is to be spared. The absence of cyanosis is accounted for by the adequate expiration of carbon dioxide, a factor which in turn undoubtedly explains the absence of dilatation of the alae nasi, seen in other obstructions in which cyanosis is present. Dilatation of the alae nasi probably results fr... medullary stimulation in the presence of ... cessive blood carbon dioxide in expiratory ins... ficiency. There is a tendency for the infant ... extend the head in order to facilitate inspirati...

Nutritional Disturbances.—The infant d... not have sufficient time to suckle or feed a... also breathe. Owing to the inadequate int... of essential and accessory foods, various ma...

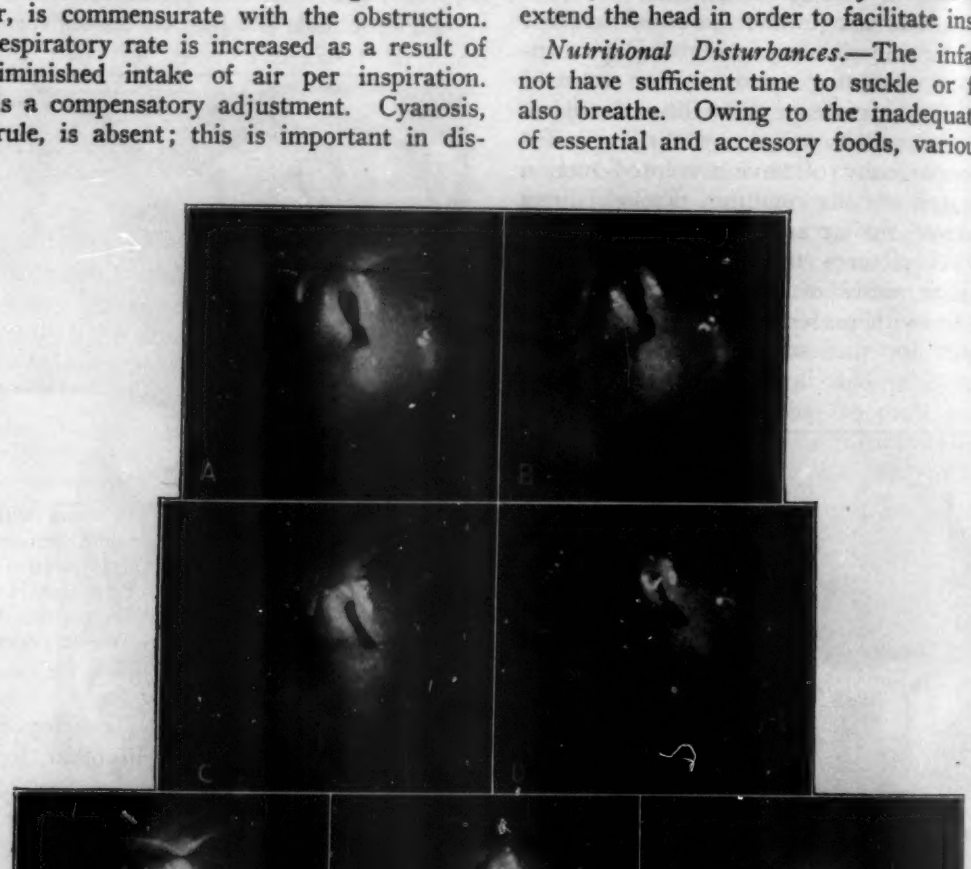

Fig. 5 (case 3).—Serial photographs demonstrating the collapse observed laryngoscopically in a case ... inspiratory laryngeal collapse. *A* to *G* show the changes observed in a single inspiration. The abnormal ch... acter of a larynx is seen in *A*. To the trained observer an accentuation of the infantile larynx is evident. T... epiglottic edges and aryepiglottic folds are in closer than normal apposition. The laryngeal introitus is reduc... in caliber to the point whereby the soft and elastic tissues forming the introital rim are collapsed by the ins... ratory intratracheal negative pressure. The photographs are serial excerpts from a Kodachrome motion pict... taken through a Flagg laryngoscope with a telephoto lens. No auxiliary lighting was necessary.

festations of nutritional deficiency are encou... tered.

DESCRIPTION OF LARYNGEAL PHENOMENA

In the typical case of inspiratory laryngeal c... lapse there is seen what has been called ... accentuation of the larynx of the infant (fig. 5... By this is meant a narrowing of the laryng... inlet by the excessively close approximation

aryepiglottic folds and lateral margins or s of the epiglottis so that the omega shape is reme. The larynx has been said to be ngated. On expiration, although the upper yngeal rim or introitus is reduced in size to ow normal, it is adequate for exhalation, and positive expiratory pressure separates the t tissues forming the rim of the introitus. nsequently there is no obstruction to expira-n. On inspiration, however, the edges of abnormally curled epiglottis are seen to proximate more closely than normal until they in complete apposition both superiorly and rsally. Almost simultaneously the short ary-iglottic folds and flaccid tissues on the summit the arytenoid eminences are drawn or sucked to the laryngeal vestibule. There follows at the d of inspiration a flattening of the posterior nvexity of the postcricoidal midline (fig. 5 *G*), s area apparently being the most yielding or astic portion of the lower laryngeal or cricoidal n. This flattening will be found to occur syn-ronously with the indrawings of the xiphoid ocess, intercostal spaces and suprasternal tch, and it takes place at that moment when spiratory obstruction or collapse is complete, e., when intratracheal and intralaryngeal pres-re is greatest.

PREVAILING THEORIES

Varied are the theories advanced to explain ese phenomena. The only ones worthy of con-deration, however, attempt to ascribe the con-tion to abnormal and excessive elasticity or abbiness of the epiglottis and other laryngeal ssues, structures which are conceded to be tremely pliable even in the normal infant. seems unreasonable that there is any appreciа-e variation of pliability or rigidity of tissues infants' larynxes. One must surely look rther for the true explanation and I am in cord with Hill,[7] who stated: "There must be me local condition, some variation or congenital alformation in the structures of this portion of e larynx which is the essential factor in at ast a majority of these cases." Chevalier ackson stated that such phenomena were often sociated with the "atypical child" but did not tate in what manner the child was atypical. oth these views are consistent with my thesis.

AUTHOR'S CONCEPTION

It is no mere coincidence, I believe, that in cases of this malady recently observed and another previously reported by me,[8] as well as in numerous cases described in medical literature, definite arrest of development of the first or mandibular arch was evident. This arrest was manifested by micrognathia or receding chin in all, and in 1 case (case 1 in my previous report[8]) by cleft palate as well. True it is that the overwhelming manifestations of obstructive laryngeal dyspnea might have diverted attention from the less impressive recession of the mandible in the other cases (cases 2 and 3; figs. 3, 4 and 10) but for previous thought and experience on the subject which directed my attention to the chin as suggesting a plausible and rational explanation of this puzzling condition.

Convinced that micrognathia of a major or even a minor degree is the primary cause underlying the condition in a large proportion of cases, I offer what I believe to be a sound anatomic basis on which to explain the syndrome as originally sought by Lees and Sutherland and by Lack.[9]

I reported on an extreme case of micrognathia in 1940[8] (fig. 6). Owing to the moribund condition of the infant, the larynx was not directly examined. It was assumed that the obstructive laryngeal dyspnea was secondary to glossoptosis as described by Robin,[10] which in turn was attributed to developmental arrest of the mandible. In retrospect I am convinced that this case qualifies as and differs nowise from a case of extreme inspiratory laryngeal collapse; no doubt direct laryngoscopy would have revealed the same phenomena heretofore described in this article. There is little doubt that the obstruction was in the laryngeal introitus and was not caused by the tongue dropping backward against the posterior hypopharyngeal wall as Robin stated.

7. Hill, F. T.: Congenital Laryngeal Stridor, Laryngoscope **40**:44, 1930.

8. Schwartz, L.: Ateliosis of the Mandibular Arch: Critical Comment on Glossoptosis, the Syndrome of Pierre Robin, Arch. Otolaryng. **31**:491 (March) 1940.

9. Inasmuch as micrognathia in turn has been shown to be but a single one of the many manifestations of developmental defect of the mandibular arch,[8] the entire syndrome under consideration should be classified as another manifestation of that developmental anomaly. In his discussion of G. B. New's paper (Congenital Obstruction of the Larynx and Pharynx, J. A. M. A. **81**:363 [Aug. 4] 1923) in which New attributed cases to "flaccid tongue" among other causes, Carmody stated: "I have seen one case of flaccid tongue which might possibly have caused obstruction, but it was a cleft palate case. . . ." This is cited as a case of "congenital laryngeal stridor" associated with a definite manifestation of developmental arrest of the mandibular arch (cleft palate). One wonders if some evidence of micrognathia might also have been present.

10. Robin, P.: La glossoptose, un grave danger pour enfants, Paris, Gaston Doin, 1929.

Summing up the situation, I believe that inspiratory laryngeal collapse (congenital laryngeal stridor) in many cases at least is due to obvious or overlooked micrognathia; that the obstructive breathing observed in "glossoptose" [10] or "ateliosis of the mandibular arch" [8] is in reality one and the same with that observed in inspiratory laryngeal collapse and should be so recognized.[11]

ANATOMIC BASIS OF CONCEPTION

A study of the epiglottis reveals the fact that several opposing forces are exerted on it (figs. 7 and 8). These forces are of two types:

1. *Muscular and Ligamentous Forces.*—(*a*) Introital Openers: These exert traction on the epiglottis from the symphysis menti as a relatively fixed point. They tend to keep the lumen of the glottis open by holding the epiglottis forward and upward and the epiglottic rim later-
ward. They apply guying action or traction
the epiglottis as follows: directly through mei
and lateral glossoepiglottic folds and contain
ligaments and through pharyngoepiglottic fol
indirectly through traction on the tongue,
hyoid bone and the hypoepiglottic ligament
the action of the geniohyoglossus muscles,
geniohyoid muscles and the mylohyoid musc

The relatively fixed point from which all th
forces operate is the symphysis menti. Wh
the symphysis is advanced forward, traction
increased and the laryngeal introitus opens
hence prognathia tends to increase traction a
open the glottic lumen whereas retrognathia
micrognathia tends to relax or to decrease t
forward traction, thereby reducing the lumen
the introitus by allowing the epiglottis to dr
downward and the edges of the epiglottis
approximate.

(*b*) Introital Closers: These exert backwa
traction from or toward the arytenoid cartila
as a relatively fixed point. They apply ba
ward and downward traction on the epiglot
tending to reduce or close the glottic lume
The pull exerted on the lateral edges brin
them into apposition. The aryepiglottic folds a
muscles (fig. 7) exert backward traction fr
the arytenoid cartilages. The thyroepiglot
muscles (fig. 8, blocked inset) pull the epiglot
downward and the edges inward as do the a
epiglottic folds and muscles. The direction
the pull is toward the arytenoid cartilage.

The equalization of these opposing forces as
occurs normally is not maintained in the micro
nathic infant. Instead, the symphysis menti
retrodisplaced; the tongue is ptosed as is t
epiglottis; traction on the hyoid bone a
the hyoepiglottic ligament is lost. The pull of t
various muscles exerting forward and late
traction on the epiglottis is relaxed, which allo
overplay of the opposing forces that exe
downward and inward pull on the epiglottis a
its lateral margins. This results in the narro
ing of the laryngeal introitus observed end
scopically.

2. *Respiratory Force.*—There is yet anoth
important force at play which further occlud
this constricted laryngeal vestibule; this is t
intralaryngeal negative pressure produced
the bellows action of the diaphragm and by t
accessory respiratory muscles. The soft tissu
of the laryngeal introitus are indrawn by t
intralaryngeal negative pressure accumulated
a result of the reduction in caliber of the lary
geal opening below its threshold of resistan

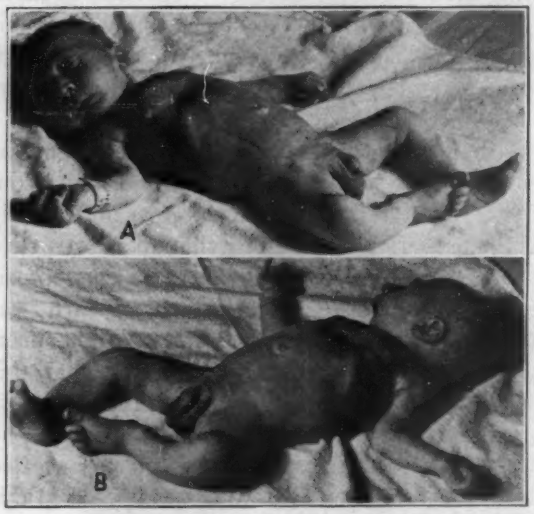

Fig. 6 (case 1).—Case reported on as illustrative of "ateliosis of the mandibular arch" by the author in 1940.[8] *A*, marked retraction of the xiphoid process characteristic of obstructive laryngeal dyspnea. *B*, micrognathism. The retraction of the xiphoid process and the intercostal spaces is well marked. The larynx was not directly inspected in this case. Nevertheless, in all clinical aspects the condition shown qualifies as a classic example of profound inspiratory laryngeal collapse ("congenital laryngeal stridor"). The micrognathia in such a case draws attention to itself; in less extreme micrognathia the obstructive laryngeal dyspnea might cause attention to be diverted from the less impressive recession of the mandible. (See figs. 3 and 4.)

11. I prefer the use of "ateliosis of the mandibular arch" to "glossoptosis" or "micrognathia" for the reason that the first title indicates a primary or major condition while the other two designate results of that condition. "Inspiratory laryngeal collapse" is also preferred to "congenital laryngeal stridor," for reasons previously cited.

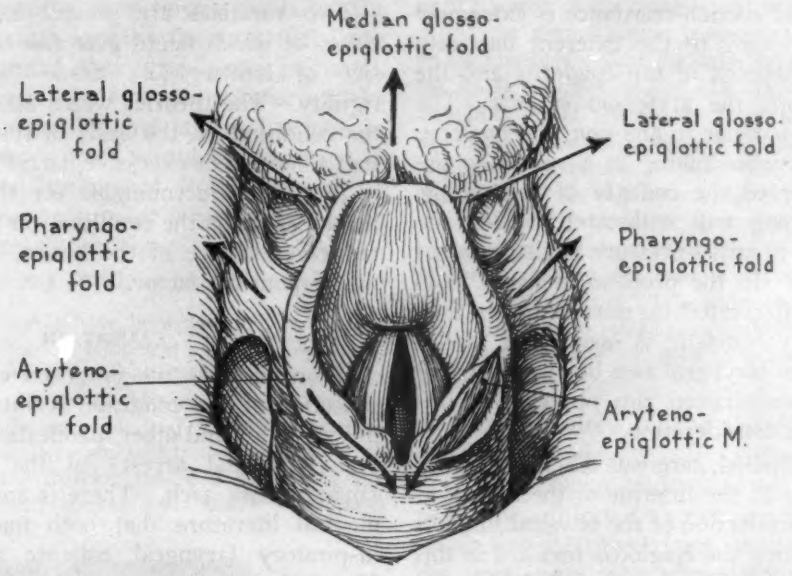

Fig. 7.—Anatomic drawing showing the superficial forces exerted on the epiglottis. Arrows point in the ection of pull. The epiglottis is maintained in a forward position, with its edges held apart, by the median ssoepiglottic, lateral glossoepiglottic and pharyngoepiglottic folds and ligaments; the epiglottis is thereby pre- nted from dropping backward and the edges from curling inward. These forces maintain a patent airway or en glottis. The opposing forces are exerted by the aryepiglottic muscles and folds and the thyroepiglottic muscles. ee inset in fig. 8.)

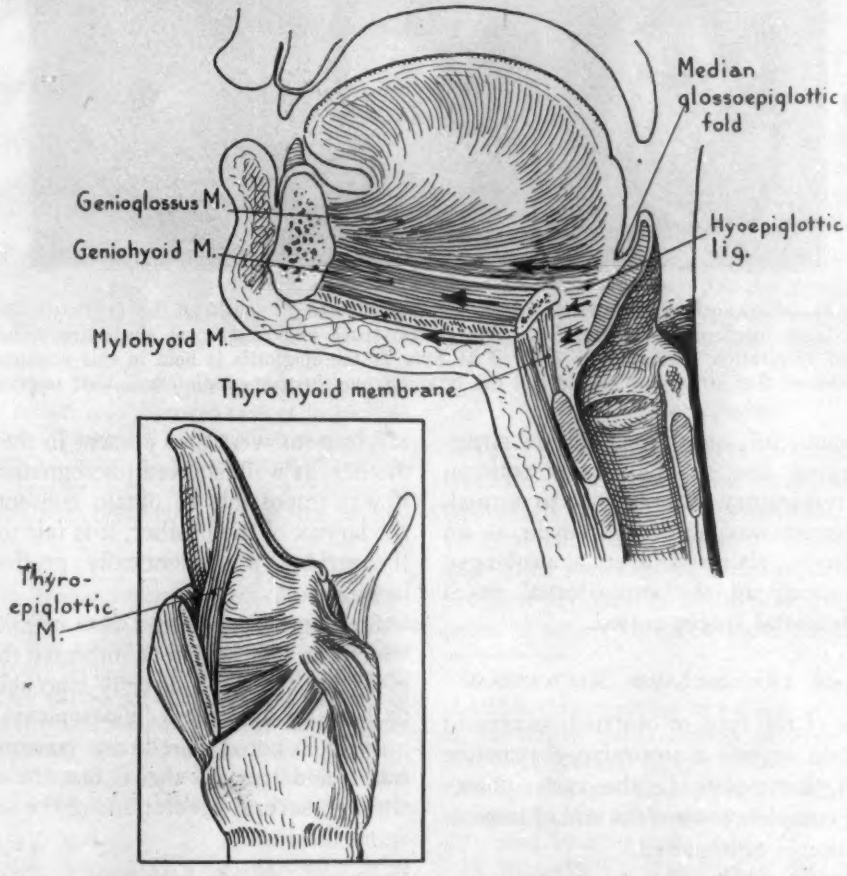

Fig. 8.—Drawing to illustrate the deep forces exerted on the epiglottis. Arrows show the direction of pull. he forward traction on the epiglottis is maintained either directly or indirectly, but entirely, from the symphysis enti as a fixed point. Retrodisplacement (micrognathia) decreases traction and permits overplay on the oppos- g forces which pull the epiglottis backward and the edges into apposition, thereby occluding the laryngeal troitus; this is what occurs in inspiratory laryngeal collapse. The inset shows the thyroepiglottic muscle. This d the aryepiglottic muscle are glottic closers. (See fig. 7 and text.)

This threshold of suction-resistance is extremely low in infants owing to the inherent flaccidity of the elastic cartilage of the epiglottis and the cartilages forming the arytenoid summit. The phenomenon is familiar to any one who has connected pliable rubber tubing to a suction apparatus and observed the collapse of the tubing. More rigid tubing will withstand the suction. The amount of negative pressure is also a factor in the equation. In the problem at hand, however, the suction created by normal inspiration, while reasonably constant, is insufficient to collapse the normal laryngeal rim but is capable of collapsing the constricted rim observed in the condition under consideration.

In the cases studied, care was taken to exclude any abnormality of the interior of the larynx as a factor in the production of the physical findings (fig. 9). In lifting the epiglottis forward in this case so as to inspect the interior of the larynx, the relief was so gratifying to the infant that

Two variables are present in this equati either of which might give rise to collapse: (size of lumen; (2) tissue "starchiness" rigidity. The theories which attempt to exp the condition on the basis of the latter cont that abnormal or excessive flaccidity of the tiss is to be held accountable for the defect. theory explains the condition on the basis of reduction in size of the lumen, which I fee the responsible factor.

CAUSATION

If one accepts the proposed explanation, causation of this condition is linked with that micrognathia and other manifestations of atelio (developmental arrest) of the first branch (mandibular) arch. There is ample evidence medical literature that both micrognathia a inspiratory laryngeal collapse are hereditar Illustrative of this is the fact that in case herein reported, micrognathia and stridor

Fig. 9 (case 3).—Exposure of the interior of the larynx obtained by pushing the epiglottis forward, there breaking the collapse mechanism (*A* and *B*). The glottic space and true vocal cords are seen to be entir normal (*C*), and respiration ceases to be labored as long as the epiglottis is held in this position. In fact, infant was so relieved that struggling ceased and the laryngoscopic instrumentation was well tolerated.

respiration became full and normal and the struggling and crying due to the instrumentation ceased. The respiratory rate returned to normal. The funnel breast was noted to remain as an established defect, acquired over a prolonged period. Indrawing of the suprasternal notch and of the intercostal spaces ceased.

DYNAMICS OF THE COLLAPSE MECHANISM

An analysis of the type of obstruction present in this condition reveals a stopvalve obstruction to inspiration, incomplete in the early phases and becoming complete toward the end of inspiration. Expiration is unimpaired.

The laryngeal introitus or inlet in the infant with inspiratory laryngeal collapse is reduced in caliber to a degree whereby the negative endolaryngeal pressure produced on inspiration overcomes the suction-resistance of the laryngeal rim.

inspirations were also present in the brother; t mother, as well, showed micrognathia. Althou it was impossible to obtain consent to exami the larynx of the brother, it is fair to assume th the stridor was identically produced in bo instances.

La Page [12] reported a case of micrognathia which the family history indicated that the deat of 3 children were directly traceable to micro nathia and associated "glossoptosis."

Pfaff [13] noted hereditary transmission in cases and traced the defect of micrognath through several generations of the same family some instances.

12. La Page, C. P.: Micrognathia in the New-Bo Lancet **1**:323, 1937.

13. Pfaff, cited by Bruning, H., and Schwalbe, Handbuch der allgemeinen Pathologie und der pat logischen Anatomie der Kindesalters, Wiesbaden, J. Bergmann, 1913, vol. 2, p. 442.

Robin's conclusion that micrognathia is the ult of congenital syphilis deserves little cre- nce. He claimed that Treponema pallidum, ving a predilection for embryonic tissue, attacks e developing mandible and arrests its growth. her investigators have found no evidence to pport this conclusion and Robin offered no equate evidence to support what appears to be more than an assumption.

Quoting from my previous publication:[14]

A variety of theories have been advanced to explain mechanism of development of micrognathia. rmally, the developing mandibular arch fuses in the dline, forming the symphysis menti. The ventral rtion becomes the mandible and the dorsal portion maxillary process, from which two shelves grow dially, fusing and forming the hard palate and the or of the nose. Keibel and Mall[15] attributed

Fig. 10 (case 2).—The middle photograph (*B*) shows mandibular recession in a proved case of inspiratory yngeal collapse ("congenital laryngeal stridor"). The milial tendency is shown by the presence of micrognathia in the brother (*A*), who had a history of idorous obstructive breathing, and in the mother (*C*).

crognathia to simple arrest in this developmental ocess. They called attention to the fact that the fetal per lip at the fifth month projects much further forard than the lower lip and suggested that an inhibitory ocess at a later period prevents outward growth of e lower portion which would overcome the disparity projection between the upper and lower portions. vis and Dunn[16] called attention to the fact that ring uterine life the entire weight of the fetus rests on the vertex of the cranium and that during the same period the sternal region transmits a distinct pressure on the mandible. Since the latter develops and unites in the midline before the maxilla, pressure of sufficient magnitude could conceivably force the partly calcified mandible backward against the softer maxilla, with consequent production of deformity. The concomitant cleft palate in the case here reported [case 1 in the present publication], in the 4 cases reported by Eley and Farber,[17] in 2 of 3 cases reported by Lannelongue and Ménard,[18] and in the case of Davis and Dunn lend weight to this theory. It is quite conceivable that sufficient pressure to retard the development of the mandible would likewise be able to prevent fusion of the palate in the midline.

The latter refers to the development of cleft palate. Conceivably, too, a normal-sized fetus developing in an undersized or infantile uterus would be subjected to extreme flexion with the result that excessive force would be exerted on the developing structures wedged between the vertex and the sternum—i. e., the maxilla and the mandible.

PROGNOSIS

It is unsafe to assume that every afflicted infant will outgrow the condition at about the age of 2 years, albeit in most cases, which are the ones of less severe underdevelopment, this is true. In cases of severe laryngeal collapse, evidenced by marked inspiratory obstruction, a guarded prognosis is warranted since many fatal cases have been encountered. There is ever present the danger of aspirational complications. Nutrition is greatly interfered with. As the infant grows older the prognosis becomes increasingly better owing to further mandibular development and consequent increased traction.

TREATMENT

The reader's attention is directed to numerous treatises dealing with the correction of micrognathia by resort to orthopedic, orthodontic, surgical and other means.[19] Undoubtedly these and other methods of dealing with this difficult problem are worthy of further study, but in the cases of severe dyspnea more direct and drastic

14. Schwartz,[8] p. 3.

15. Keibel, F. K. J., and Mall, F. P.: Manual of man Embryology, Philadelphia, J. B. Lippincott mpany, 1910.

16. Davis, A. D., and Dunn, R.: Micrognathia: A ggested Treatment for Correction in Early Infancy, J. Dis. Child. **45**:799 (April) 1933.

17. Eley, R. C., and Farber, S.: Hypoplasia of the Mandible (Micrognathy) as a Cause of Cyanotic Attacks in the Newly Born: Report of Four Cases, Am. J. Dis. Child. **39**:1167 (June) 1930.

18. Lannelongue, O. M., and Ménard, V.: Affections congenitales, Paris, Asselin & Houzeau, 1891, vol. 1, p. 423; cited by Eley and Farber.[17]

19. Babcock, W.: The Surgical Treatment of Certain Deformities of the Jaw Associated with Malocclusion of the Teeth, J. A. M. A. **53**:833 (Sept. 11) 1909. Hensel, G. C.: The Surgical Correction of Mandibular Protraction, Retraction and Fractures of Ascending Rami, Surgery **2**:92, 1937. Davis and Dunn.[16]

measures applied to the site of obstruction are demanded if life is to be spared.

Two such immediate remedial measures are worthy of consideration when obstructive laryngeal dyspnea is profound and threatens the life of the infant. One is tracheotomy. The other is partial epiglottidectomy.

Partial epiglottidectomy apparently is not fraught with the grave risk that is generally believed to accompany this procedure. In case 3 I removed a small V-shaped wedge from the free edge of the epiglottis in the midline with no untoward results and with apparently little discomfort to the infant. This was accomplished through an infant laryngoscope and laryngeal forceps which removes such a V-shaped wedge. There was no appreciable bleeding, and no anesthesia was required. The child's condition improved immediately, and subsequently he appeared permanently relieved of his obstructed breathing. Where relief is not obtained readily, consideration should be given to removal of an even greater portion of the free edge by amputation. Some such procedure undoubtedly will be developed which will be the method of choice. The ideal approach will be the one which breaks the obstructive mechanism while disturbing the normal physiology of the airway to the least possible degree. This approach remains to be developed.

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BLAST INJURY OF THE EARS

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AND
CAPTAIN H. P. SCHENCK (MC), U.S.N.R.

In this presentation we shall describe the rious types of injury of the ear resulting from st waves, discuss the causative factors, show e audiometric findings and give a brief descrip- n of the treatment employed by us.

Of 3,450 casualties admitted to a hospital ship ring the first few months of the Solomon ands Campaign, 1,922 were classified as hav- traumatic or surgical injuries, and 82 2 + per cent) of the latter group had blast ury of the ears serious enough to warrant servation and treatment by the otolaryngol- ist. Many patients had varying degrees of ral damage from concussion waves, but blast uries of a more serious character to the tho- ic or the abdominal viscera of these patients cluded extensive otologic study, and for this son they were not included in this series.

Many facts of theoretic and practical appli- ion were obtained from this study, but for sons beyond our control their presentation necessarily limited in scope. Patients were t on board the hospital ship for only brief riods during active evacuations. Follow-up ta on hearing acuity and on discharges from e ears are lacking. Some of the hearing levels e inaccurate because of ship's noises, lack of undproof facilities or inability of certain tients who had been exposed to intense blast aves to concentrate during the audiometric tests.

TYPES OF INJURY

In this series of patients the commonest type aural lesion encountered was traumatic rup- e of the tympanic membrane with or without condary infection. Other forms of aural mage included traumatic deafness, which was most universally present in varying degree, emorrhage into the middle ear cavity, dislocа- on of the ossicles and labyrinthine hemorrhage.

Acoustic trauma in modern warfare is gen- ally produced by blast waves from the detona- on of high explosives or by repeated or con- nuous exposure to excessive sound stimuli. he precipitating causes for aural damage in this roup of patients were: (*a*) atmospheric blast rising from the detonation of aerial bombs, large liber shells, mortar shells, hand grenades and nd mines and (*b*) immersion blast from deto- nation of depth charges when the subject was in the water.

Various factors of importance in the causation of acoustic trauma and the resultant deafness in service personnel are: (1) the proximity to the source of the sound or the center of detonation, (2) the character of the concussion waves (whether atmospheric or immersion blast), (3) the duration of the exposure to the sound stimulus, (4) the awareness of the subject that the blast is impending, (5) the presence or the absence of previous aural disease and (6) the use of protective devices.

Blast waves from detonation of high explosives have a profound effect on the auditory apparatus of a person who is within the critical distance of the center of detonation. This has been estimated to be about 20 feet (6 meters) in air and approximately four times that distance in water.

INJURY DUE TO ATMOSPHERIC BLASTS

Atmospheric blast waves have been described [1] as excessively intense longitudinal sound waves with at least two components: a compression phase, lasting approximately 0.005 second, and a suction phase, lasting about twice as long. The pressure exerted by the first phase varies inversely with the square of the distance and reaches maximum intensity in less than 1 millisecond. The magnitude of both components is directly proportional to the size of the charge used, and the resultant damage to the ears may be slight or severe, depending on the intensity and the proximity of the blast waves.

The reflex response of the tensor tympani and stapedius muscles tends to protect the internal ear against sudden excessive sound stimuli. The latent period of this reflex is between 5 and 10 milliseconds, and consequently the reflex is of no value as a protective mechanism against blast injury.

In blast injury of the ear the degree of deafness is usually greater when the tympanic membrane remains intact than when it is ruptured, provided each exposure to blast is of comparable

1. Greaves, F. C.; Draeger, R. H.; Brines, O. A.; Shaver, J. S., and Corey, E. L.: An Experimental Study of Underwater Concussion, U. S. Nav. M. Bull. **41**:339 (March) 1943.

intensity. It is thought that much of the initial concussion wave is dissipated when the drum ruptures and that in this way the cochlear structures are protected.[2] In patients subjected to intense blast waves, which are intensified to a greater degree in the narrow external auditory canal, the full impact of the pressure is transmitted to the endolymph of the internal ear if the drum does not rupture, with resulting disintegration of cells of the organ of Corti. Although evidence of hemorrhage into the internal ear or the labyrinth is not common, some of the patients complained of persistent dizziness with demonstrable nystagmus. This was usually associated with other signs of concussion which included a period of unconsciousness, hemoptysis and inability to concentrate.

Blurring of vision was a common complaint following blast injury, and in most of the patients with this complaint the usual findings of hyperemia and slight elevation of the disk and blurring of the margins were evident on ophthalmoscopic examination. Over half of the cases had injuries of other parts of the body from blast waves, shrapnel or bullets.

INJURY FROM IMMERSION BLASTS

"Immersion blast" is the term given[3] to injuries sustained as a result of detonation of high explosives under water. One patient in this series (case 7, table 1) stated that his head was under water when his ship blew up 20 feet from him. As a result of this immersion blast, his audiometric curve (chart 1) showed an average of 86 decibels of loss in his right ear and 95 decibels of loss in his left ear (the side nearer the explosion) with no demonstrable bone conduction on either side.

This is a total loss of serviceable hearing, and his prospects of recovery are minimal inasmuch as the audiometric studies were made two weeks after his injury. In our experience the maximum recovery of auditory acuity following blast occurs within this interval. Tinnitus usually disappears early if there is no serious damage to the internal ear. The persistent type of tinnitus, which was present in 16 patients in this series, is high pitched, constant and bilateral, and the associated audiometric curve shows a predominant loss in the higher frequencies.

INJURY FROM REPEATED EXCESSIVE SOUND STIMULI

Exposure to excessive sound stimuli results in damage which differs from that induced by blast waves. In the majority of the audiograms patients subjected to blast the greatest loss was the higher frequencies, but the tonal dip at 4 cycles seen so commonly in cochlear damage to industrial noises and repeated excessive sou stimuli was not evident in many of these aud grams. There was a relatively larger dec loss in patients with secondary infection, due the associated impairment of conduction.

It has been observed that the total time exposure to a loud sound is of major importa in determining the extent of the damage to cochlear mechanism and whether this dama is to be temporary or permanent. Short ex sures to sound of moderate intensity result temporary impairment of auditory acuity. T has been described as auditory fatigue. If duration of exposure is extended, the proc changes from one of fatigue to permanent de ness of varying degrees. This permanent char is shown in chart 2.

If the intensity of the sound or of the bl waves giving rise to acoustic trauma is of su cient magnitude, only a brief exposure may pr duce severe cochlear changes and consequ profound deafness, which usually is irreversib The following case history illustrates this poi

CASE 62.—A 46 year old man in the service admitted to this hospital ship complaining of bilate deafness and persistent tinnitus. Six weeks be admission he was standing 15 feet below a 5 i gun turret on his ship when the gun was fired with warning to him. Immediately following this bilate tinnitus and deafness developed, worse in his right e There was no history of rupture of his tymp membranes or of previous aural disease. Examinat revealed retracted drums; the Rinne test was posit on both sides, and there was marked shortening of and bone conduction. An air conduction audiogr (chart 3) with masker revealed an average loss in frequencies of 68 decibels in his right ear and decibels in his left ear, with the greatest loss 2896 cycles.

It is assumed that the protection afforded the patient in case 5 by the turret structu eliminated most of the pressure waves from t blast of the 5 inch gun and that the hearin defect was produced by repeated intense sou stimuli, whereas the patient in case 62, with o exposure to the blast waves from a simil detonation, sustained a severe hearing lo throughout the audiometric range.

SUMMARY OF CASES (TABLES 1 AND 2)

A summary of the findings in the 82 patien studied after their exposure to blast waves found in table 1. The interval of time betwe the blast injury and our examination vari between two days and three months. The com monest cause of acoustic trauma in this grou

2. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol., Rhin. & Laryng. **50**:70 (March) 1941.

3. Palma, J., and Uldall, J. J.: Immersion Blast Injuries, U. S. Nav. M. Bull. **41**:3 (Jan.) 1943.

detonation of aerial bombs, and the usual acter of the resulting audiometric curve is vn in chart 4.

orty-one patients (50 per cent) gave a his- of exposure to blast waves from bombs, and his group 11 were unconscious following concussion. Fifty-eight patients (70.7 + cent) had perforation of the tympanic mem- e; in 33 this was unilateral and in 25 was bilateral. Of those with unilateral per- ation, 18 had it on the right side and 15 on left side. Bila eral perforations were caused ncipally by aerial bombs, depth charges and mortar shell explosions. The types of detona- tion causing auditory trauma are shown in table 2.

There was wide variation in the findings from audiometric studies and tuning fork tests in patients subjected to blast waves. In large measure this variation was directly proportional to the distance from the center of detonation. One patient (case 10) had a normal audiogram one week after being exposed to the blast effect of a depth charge. He was about 80 feet from the center of detonation, which in water is the upper limit of the so-called "critical distance,"

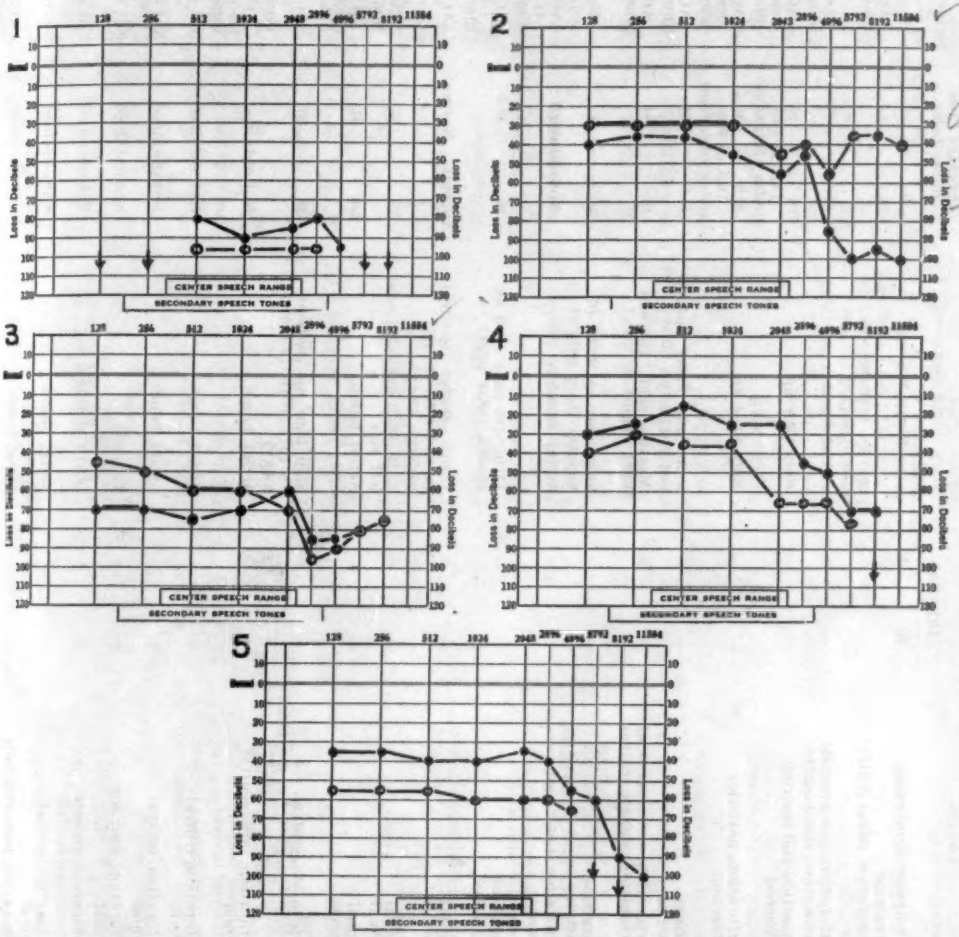

Chart 1.—Immersion blast from explosion of a ship. The patient (case 7, table 1) was 20 feet from the ter of detonation, his head under water. The audiogram was made two weeks after the injury. The blank les represent the threshold of hearing in the left ear; the black ones, that in the right ear.

Chart 2.—Excessive sound stimuli. The patient (case 5) was exposed to repeated firing of a 5 inch gun ing a sea battle. He was inside a gun turret, protected from blast waves. The audiogram was made four ks after the injury.

Chart 3.—Detonation of a 5 inch gun. The patient (case 62) was exposed to one explosion while 15 feet n the center of detonation without protection. The audiogram was made six weeks after the injury.

Chart 4.—Aerial bomb explosion. The subject (case 2) was 20 feet from the center of detonation. He was unconscious afterward. Both tympanic membranes were ruptured. The audiogram was made two weeks r the injury.

Chart 5.—Mortar shell explosion. The subject (case 6) was 10 feet away. He was unconscious for thirty utes. Both tympanic membranes were ruptured. The audiogram was made two weeks after the injury.

TABLE 1.—*Condensed Clinical Findings in Patients with Blast Injury of Ears*

Case	Age	Complaint	Tympanic Membranes	Infection R	Infection L	Blast Waves	Audiometer (Average Loss in Decibels) R	Audiometer (Average Loss in Decibels) L	Tuning Fork Tests R	Tuning Fork Tests L	Interval Between Injury and Examination
1	26	Bilateral tinnitus; bilateral deafness	R—Retraction L—Perforation, linear central	—	—	5 in. (12.5 cm.) shell exploded 3 ft. (91 cm.) from patient	70.5	44	AC > BC * (diminished)	AC > BC (diminished)	5 wk.
2	20	Bilateral tinnitus; discharge from left ear	R—Perforation, small central L—Perforation, small central	+	+	Aerial bomb	55	72	AC > BC (diminished)	AC > BC (diminished)	2 wk.
3	24	Bilateral tinnitus	R—Retraction and scarring L—Thickened	—	—	Ship blew up	28 Dip. at 4096 double vibrations	25	AC > BC (slightly diminished)	AC > BC (slightly diminished)	2 wk.
4	19	Bilateral deafness	R—Perforation, posterior L—Retraction	—	—	Ship blew up	23.5 Dip. at 4096 double vibrations	18.5	AC > BC (slightly diminished)	AC > BC (slightly diminished)	17 days
5	25	Bilateral tinnitus; bilateral deafness	R—Normal L—Scarring	—	—	5 in. gun firing during sea battle (no blast effect)	44.5	52	AC > BC (diminished)	AC > BC (diminished)	4 wk.
6	22	Bilateral aural discharge; bilateral deafness	R—Perforation, stellate central L—Perforation, stellate central	+	+	Mortar shell (patient unconscious 30 min.)	48	84	AC > BC (diminished)	AC > BC (markedly diminished)	2 wk.
7	24	Bilateral deafness	R—Retraction L—Scarring	—	—	Ship blew up 20 ft. (6 meters) from patient	86	95	No bone conduction	No bone conduction	2 wk.
8	20	Bilateral deafness	R—Perforation, lower half L—Normal	—	—	Aerial bomb	Not obtained		AC > BC (slightly diminished)	AC > BC (moderately diminished)	3 wk.
9	22	Bilateral tinnitus; dizziness	R—Retraction L—Retraction	—	—	15 in. (38 cm.) shell exploded (near miss)	38	32.5	AC > BC (slightly diminished)	AC > BC (slightly diminished)	4 wk.
10	19	Blurred vision	R—Normal L—Normal	—	—	Depth charge (patient in water 80 ft. [24 meters] away)	Normal	Normal	AC > BC N	AC > BC N	1 wk.
11	23	Deafness, left ear; blurred vision	R—Normal L—Normal	—	—	15 in. shell exploded near left side	28	35	(Slightly diminished)	(Slightly diminished)	2 wk.
12	21	Bilateral deafness; headache	R—Normal L—Normal	—	—	Aerial bomb	36	41	AC > BC (moderately diminished)	AC > BC (moderately diminished)	2 wk.
13	19	Bilateral deafness; discharge from right ear	R—Perforation, central L—Perforation, posterior marginal	+	—	Ship blew up (patient on raft 30 ft. [9 meters] away)	20	20	AC > BC (both diminished)	AC > BC (both diminished)	1 wk.
14	23	Deafness on right	R—Perforation, large central L—Normal	—	—	Aerial bomb	Not obtained		AC > BC (moderately diminished)	AC > BC (slightly diminished)	5 days
15	22	Deafness on right; discharge from right ear	R—Perforation, central L—Normal	+	—	Aerial bomb	Not obtained		Not obtained	Not obtained	1 wk.
16	20	Deafness on right; discharge from right ear	R—Perforation, central L—Normal	+	—	Aerial bomb	Not obtained		Not obtained	Not obtained	1 wk.
17	34	Deafness on right; discharge from right ear	R—Perforation, anterior L—Normal	+	—	Torpedo	Not obtained		Not obtained	Not obtained	9 days
18	23	Bilateral deafness	R—Perforation, stellate L—Perforation, stellate	+	+	Depth charge	Not obtained		Not obtained	Not obtained	2 wk.
19	22	Deafness on right	R—Perforation, stellate L—Normal	—	—	Aerial bomb	Not obtained		Not obtained	Not obtained	10 days
20	20	Deafness on right; discharge from right ear	R—Perforation, posterior half L—Retracted	+	—	Aerial bomb	Not obtained		AC > BC (moderately diminished)	Not obtained	1 wk.
21	20	Bilateral deafness; discharge from left ear	R—Normal L—Perforation, anterior, red and thickened	—	+	Aerial bomb exploded 15 ft. (4.5 meters) away	Not obtained		AC > BC (slightly diminished)	AC > BC (moderately diminished)	2 wk.

21	20	Bilateral deafness; discharge from left ear	R—Normal L—Perforation, anterior, red and thickened	—	+	Aerial bomb exploded 15 ft. (4.5 meters) away	Not obtained		AC > BC (slightly diminished)	AC > BC (moderately diminished)	2 wk.
22	22	Bilateral deafness	R—Normal L—Perforation, anterior marginal	—	—	Aerial bomb	Not obtained		Not obtained	Not obtained	2 mo.
23	23	Bilateral deafness; discharge from left ear	R—Perforation, anterior, red and thickened L—Perforation, anterior half gone	+	—	Aerial bomb exploded at 20 ft. (6 meters) (patient unconscious 5 min.)	45	40	AC = BC (moderately diminished)	AC = BC (moderately diminished)	2 wk.
24	20	Deafness on left; discharge from left ear	R—Normal L—Perforation, central	—	+	Aerial bomb	35	65	AC > BC (moderately diminished)	AC > BC (moderately diminished)	2 wk.
25	23	Bilateral deafness; bilateral discharge	R—Perforation, central stellate L—Perforation, central stellate	+	+	Hand grenade	60	60	Not obtained	Not obtained	12 days
26	28	Bilateral deafness; bilateral discharge	R—Perforation, central stellate L—Perforation, central stellate	+	+	Depth charge	Not obtained		Not obtained	Not obtained	1 wk.
27	24	Bilateral deafness; bilateral discharge	R—Perforation, central stellate L—Perforation, central stellate	+	+	Torpedo	42	48	Not obtained	Not obtained	1 wk.
28	23	Deafness on right; discharge from right ear	R—Perforation, anterior L—Normal	+	—	Shell exploded in compartment with him	Not obtained		Not obtained	Not obtained	2 wk.
29	26	Deafness on right	R—Perforation, stellate L—Normal	—	—	Aerial bomb exploded 15 ft. from patient	54	28	AC = BC (markedly diminished)	AC > BC (moderately diminished)	2 wk.
30	23	Deafness on right	R—Healed perforation L—Normal	—	—	Land mine exploded (patient in jeep)	27	18.5	AC > BC (slightly diminished)	AC > BC (slightly diminished)	2 wk.
31	25	Bilateral deafness	R—Perforation, stellate, anterior L—Perforation, stellate, anterior	—	—	Mortar shell	Not obtained		Not obtained	Not obtained	1 wk.
32	26	Deafness and tinnitus on right	R—Retracted L—Retracted	—	—	8 in. (20 cm.) shell	Not obtained		AC > BC (diminished)	AC > BC (diminished)	3 wk.
33	20	Deafness on right; discharge from left ear	R—Perforation, large central L—Perforation, large central	+	—	Land mine	Not obtained		Not obtained	Not obtained	3 days
34	23	Bilateral deafness; discharge from left ear	R—Perforation, anterior half gone L—Perforation, inferior marginal	—	+	Aerial bomb 6 ft. (about 2 meters) from him	Not obtained		Not obtained	Not obtained	3 days
35	18	Bilateral deafness; bilateral discharge	R—Perforation, central stellate L—Perforation, central stellate	+	+	Aerial torpedo and depth charge	Not obtained		Not obtained	Not obtained	1 wk.
36	23	Deafness on right	R—Perforation, posterior L—Normal	—	—	Aerial bomb	55	25	Normal	Normal	1 wk.
37	24	Deafness on right; discharge from right ear	R—Perforation, posterior; drum red L—Normal	+	—	Aerial bomb	Not obtained		Not obtained	Not obtained	2 wk.
38	21	Deafness on right	R—Healed perforation, inferior L—Normal	—	—	Aerial bomb	Not obtained		AC > BC (slightly diminished)	AC > BC (slightly diminished)	3 wk.
39	25	Deafness on right	R—Perforation, anterior and inferior L—Normal	—	—	Mortar shell	Not obtained		AC > BC (moderately diminished)	AC > BC (about normal)	4 wk.
40	22	Bilateral deafness	R—Perforation, linear central L—Perforation, large anterior	—	—	Aerial bomb (patient unconscious few minutes)	48	60	Not obtained	Not obtained	3 days
41	26	Bilateral deafness	R—Retraction L—Retraction	—	—	Aerial bomb 6 ft. away (patient unconscious)	72	64	AC = BC (moderately diminished)	AC = BC (moderately diminished)	4 days
42	21	Bilateral deafness	R—Perforation, healing, anterior L—Normal	—	—	Artillery shell 15 ft. from him	70	No tones heard	Not heard by AC or BC		2 wk.
43	22	Bilateral deafness; bilateral tinnitus	R—Perforation, stellate L—Perforation, stellate	—	—	Aerial bomb (shrapnel hit on right)	55	35	Not obtained	Not obtained	10 days
44	25	Deafness on left	R—Perforation, posterior L—Perforation, inferior	—	—	Aerial bomb (patient unconscious 1 hr.)	40	65	Not obtained	Not obtained	2 wk.

TABLE 1.—*Condensed Clinical Findings in Patients with Blast Injury of Ears—Continued*

Case	Age	Complaint	Tympanic Membranes	Infection R	Infection L	Blast Waves	Audiometer (Average Loss in Decibels) R	Audiometer (Average Loss in Decibels) L	Tuning Fork Tests R	Tuning Fork Tests L	Interval Between Injury and Examination
45	21	Bilateral deafness; discharge from left ear	R—Normal L—Perforation, central	—	+	Aerial bomb 30 ft. away (patient unconscious 30 min.)	Not obtained		Not obtained	Not obtained	2 wk.
46	19	Bilateral deafness; discharge from both ears	R—Perforation, stellate L—Perforation, stellate	+	+	Aerial bomb 12 ft. (3.5 meters) away	Not obtained		Not obtained	Not obtained	12 days
47	19	Bilateral deafness; bilateral discharge	R—Perforation, large central L—Perforation, large anterior	+	+	Aerial bomb hit his compartment	Not obtained		AC > BC (moderately diminished)	AC > BC (moderately diminished)	4 days
48	20	Deafness on left	R—Retraction L—Perforation, anterior	—	—	Aerial bomb within few feet (patient unconscious 4 min.)	40	40	Not obtained	Not obtained	2 wk.
49	24	Bilateral deafness	R—Perforation, posterior and inferior L—Perforation, posterior and inferior	—	—	Aerial bomb, same compartment, 20 ft. from patient	35	50	AC > BC (slightly diminished)	AC > BC (slightly diminished)	4 days
50	22	Bilateral deafness; bilateral discharge	R—Perforation, central L—Perforation, central	+	+	Aerial bomb (near miss)	Not obtained		Not obtained	Not obtained	3 days
51	21	Deafness and discharge on left	R—Hemorrhage over umbo L—Perforation, inferior	—	+	Aerial bomb 10 ft. (3 meters) away (patient unconscious few min.)	48	34	Not obtained	Not obtained	3 days
52	22	Bilateral deafness; bilateral discharge	R—Perforation, central L—Perforation, central; incus dislocated	+	+	Aerial bomb (face hit by bomb fragments)	65	80	Not obtained	Not obtained	10 days
53	19	Bilateral deafness	R—Marked retraction L—Marked retraction	—	—	Hand grenade 18 in. (45.5 cm.) from face (patient unconscious 10 hr.)	58	66	AC = BC (almost absent)	AC = BC (absent)	12 days
54	22	Bilateral deafness; discharge from right ear	R—Perforation, central L—Normal	+	—	Aerial bomb 15 ft. away (patient unconscious few minutes)	45	56	AC = BC (moderately diminished)	AC > BC (moderately diminished)	15 days
55	20	Deafness on right	R—Normal L—Normal	—	—	Hand grenades (shrapnel hit right mastoid region)	48	28	Not obtained	Not obtained	2 wk.
56	26	Bilateral deafness; bilateral tinnitus	R—Normal L—Normal	—	—	Aerial bomb at 15 ft.	36	34	AC > BC (moderately diminished)	AC > BC (moderately diminished)	4 wk.
57	20	Bilateral deafness	R—Normal L—Normal; drum deep red	—	—	Torpedo and depth charges	Not obtained		AC > BC (moderately diminished)	AC > BC (moderately diminished)	1 wk.
58	20	Bilateral deafness; pain in ears	R—Injected L—Injected	—	—	Depth charges (patient in wate.)	35	44	.....	.....	1 wk.
59	18	Bilateral deafness	R—Perforation, linear, central L—Perforation, linear, central	—	—	16 in. (40.5 cm.) shell exploded near (patient unconscious 30 min.)	67	72	AC = BC (markedly diminished)	AC = BC (markedly diminished)	5 days
60	18	Deafness on left	R—Normal L—Perforation, central	—	—	Aerial bomb	30	38	Not obtained	Not obtained	6 wk.
61	26	Deafness and discharge on right	R—Perforation, posterior and inferior, irregular L—Normal	+	—	"K" gun at 3 ft.	42	24	AC = BC (moderately diminished)	AC > BC (slightly diminished)	2 days

62	46	Bilateral deafness; bilateral tinnitus	R—Scarred and retracted L—Scarred and retracted	—	—	5 in. gun at 15 ft.	68	74	AC = BC (markedly diminished)	AC = BC (markedly diminished)	6 wk.
63	23	Deafness and discharge on right	R—Perforation, inferior and posterior L—Normal	+	—	Aerial bomb	Not obtained		Not obtained	Not obtained	2 wk.
64	23	Deafness and discharge on left	R—Retraction; scarring L—Perforation, upper posterior	—	+	Aerial bomb	52	36	AC > BC (moderately diminished)	AC > BC (greatly reduced)	4 wk.
65	19	Deafness and discharge on left	R—Retraction L—Perforation, central	—	+	Aerial bomb	Not obtained		Not obtained	Not obtained	6 wk.
66	20	Deafness on left	R—Normal L—Perforation, anterior, inferior	—	—	Aerial bomb	Not obtained		Not obtained	Not obtained	2 wk.
67	24	Bilateral deafness	R—Retraction and scarring L—Retraction and scarring	—	—	Depth charges	52.5	52	AC > BC (moderately diminished)	AC > BC (moderately diminished)	12 wk.
68	22	Bilateral deafness; bilateral tinnitus	R—Retraction L—Retraction	—	—	5 in. gun (patient unprotected)	28	30	AC > BC (normal)	AC > BC (normal)	8 wk.
69	26	Bilateral deafness	R—Retraction and scarring L—Retraction and scarring	—	—	5 in. gun (patient unprotected)	66.5	70	AC > BC (moderately diminished)	AC > BC (moderately diminished)	6 wk.
70	23	Bilateral deafness	R—Normal L—Normal	—	—	5 in. gun (patient unprotected)	40.5 Dip at 4096 double vibrations	45	AC > BC (moderately diminished)	AC > BC (moderately diminished)	8 wk.
71	22	Bilateral deafness; intermittent tinnitus	R—Normal L—Normal	—	—	Aerial bomb at 20 ft. (patient unconscious 30 min.)	57	57	AC > BC (moderately diminished)	AC > BC (moderately diminished)	4 wk.
72	23	Deafness on left	R—Normal L—Normal	—	—	8 in. (20 cm.) gun	30	45	AC > BC (slightly diminished)	AC > BC (moderately diminished)	3 days
73	38	Deafness on right	R—Perforation, inferior L—Normal	—	—	Aerial bomb	Not obtained		AC > BC (slightly diminished)	AC > BC (slightly diminished)	1 wk.
74	18	Bilateral deafness; bilateral tinnitus	R—Normal L—Normal	—	—	Mortar shell	80	45	AC + BC (not heard)	AC + BC (moderately diminished)	12 wk.
75	24	Bilateral deafness; bilateral tinnitus, bilateral discharge	R—Perforation, large, posterior, inferior L—Perforation, large, posterior, inferior	+	+	Ship blew up 25 ft. (7.5 meters) away	Not obtained		Not obtained	Not obtained	8 days
76	28	Deafness and discharge on left	R—Normal L—Perforation, stellate	—	+	Ship blew up 30 ft. away	Not obtained		Not obtained	Not obtained	8 days
77	21	Bilateral deafness; bilateral tinnitus	R—Perforation, linear L—Perforation, linear	—	—	16 in. shell (patient unconscious 30 min.)	Not obtained		Not obtained	Not obtained	2 days
78	23	Deafness on left	R—Normal L—Perforation, central	—	—	Aerial bomb few feet away (patient unconscious 4 min.)	Not obtained		Not obtained	Not obtained	12 days
79	20	Deafness, tinnitus and discharge, all on left	R—Normal L—Perforation, large, posterior	—	+	Mortar shell	Not obtained		Not obtained	Not obtained	8 wk.
80	25	Bilateral deafness; bilateral discharge	R—Perforation, posterior L—Perforation, central	+	+	Torpedo	Not obtained		Not obtained	Not obtained	2 days
81	21	Bilateral deafness; bilateral tinnitus; bilateral discharge	R—Perforation, stellate L—Perforation, stellate	+	+	Aerial bomb (1,200 lb.) 12 ft. away	Not obtained		Not obtained	Not obtained	11 days
82	22	Deafness on left	R—Normal L—Perforation, stellate	—	—	Aerial bomb	Not obtained		Not obtained	Not obtained	4 days

and sustained no residual damage of the ear, but had blurring of vision and the typical associated ophthalmoscopic findings. In contrast to this are the drastic changes at lesser distances from the center of detonation as illustrated in case 7, presenting the audiometric curve shown in chart 1.

Suppurative otitis media was present at the time of admission in 35 (42.6 per cent) of the 82 patients. It has been stated that infection rarely occurs in traumatic rupture of the tympanic membrane if the condition is properly managed, yet secondary infection developed in over 40 per cent of the patients with perforation in this group. Few gave a history of having had their ears irrigated, a procedure which is mentioned only to be condemned because of the danger of introducing infection into the tympanic cavity. The reason for such a high percentage

TABLE 2.—*Detonations Causing Auditory Trauma*

	Cases
Aerial bomb	41
Large caliber shell	14
Exploding ship	6
Depth charge	5
Mortar shell	5
Hand grenade	3
Torpedo	3
Aerial torpedo and depth charge	2
Land mine	2
"K" gun	1
Total	82

of secondary otitis media in this group seems to be that practically all the patients were either lying in dirty fox holes at the time of injury or were floating in water contaminated with fuel oil and debris from sinking ships. Inasmuch as infection developed in such a high proportion of the patients without the technical error of syringing, it might be prudent to suggest the prophylactic measure of applying locally microcrystals of sulfathiazole in either the powder or the suspension form as soon as possible after traumatic rupture has occurred. Another preventive measure which would be effective against both blast waves and infection is the use of rubber ear plugs especially designed for that purpose. In a controlled series studied by the British Navy these plugs were found to be very effective, but the problem of having service personnel use them is a difficult one. When obturators are being worn in the external auditory canals, it is possible for the pressure effect of the blast wave, if intense enough, to rupture the tympanic membranes by blowing them outward, the pressure in this instance coming through the eustachian tubes. Although no patients with this type injury were seen in our series, such a mech nism of injury has been described [4] with t clinical findings of everted margins of the r tured tympanic membranes rather than the us inverted margins. The simple expedient plugging the external auditory meati with cott with or without oil or wax, has been found to effective against blast waves.

TREATMENT

The treatment of blast injuries of the ear unsatisfactory. When secondary infection d not supervene, a traumatic perforation of t drum membrane heals promptly. If the bl is recent, some assurance can be given to t subject that much of his deafness and tinnit will disappear provided the exposure occurr at a distance greater than one quarter of t "critical distance" from the center of deton tion. For patients whose history and examin tion reveal that such a prognosis is warrante the reassurance itself is good treatment. Occ sionally both the hearing and the tinnitus c be improved by inflation of the eustachian tub particularly when the tympanic membrane intact. Deep diathermy, concentrated vitamin and rest are adjuvants. These patients shou not be allowed to return to duty with exposure further detonations until the recovery of t acoustic mechanism is complete. If there superimposed suppurative otitis media, the insu flation of microcrystalline sulfathiazole powd or the instillation of a suspension of this dru into the external auditory canal will prompt control the infection. In none of the patien in this series did infection of the mastoid proce develop.

The psychologic problem presented by som of these patients is a complicated one. In add tion to the suspicious and introverted behavi so commonly seen in deafened persons, many these also manifest great concern as to th prognosis of their deafness and tinnitus an show all the classic symptoms of combat fatigu or "shell shock." This difficult interval require judicious reassurance on the part of the otologi and occasionally the cooperation of the psy chiatrist.

Finally, we feel that a perusal of table 1 wi enable the reader to appreciate the diversity o the physical and audiometric findings in subject exposed to blast waves.

4. Colledge, L., and others: Injuries of the Ea A Discussion, Proc. Roy. Soc. Med. (Sect. Otol **34**:5 (Nov.) 1940.

PATHOLOGY OF PETROSITIS

REPORT OF THREE CASES

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Perhaps the subject of petrositis or apicitis is historical interest only; however, the otolo-st's interest in the pyramids of the petrous nes is not yet to be relegated to the past. ragedies of meningitis and cerebral abscess are ill to be the otologist's problem, and petrositis going to be the ally of these serious compli-tions.

In the past few months 2 cases of meningitis hich were apparently due to petrositis have me under my study. Although the temporal nes have not yet been sectioned, there is every idence clinically and on gross pathologic exam-ation that they contained the nidus of infection om which meningitis resulted as a complication. oth of the patients, it may be observed, received assive doses of the sulfonamide drugs.

Therefore, I present 3 cases which it is hoped ill offer useful data in the management of the ses of petrosis which are sure to occur.

Histologically, the petrosa may consist of bone arrow or of air cells or of a mixture of both arrow and air cells. Approximately 20 per nt of specimens contain marrow and are di-loic; 35 per cent contain pneumatic cells; the emaining 45 per cent show a mixture of mar-ow and cells.

If marrow is present, it may be highly hemo-oietic or diploic, or it may consist of fat and brous tissue with only a few areas of hemo-enesis present. The first type of myeloid tissue s usually found in the young patient. The sec-nd type is usually present in the adult patient.

If such a bone becomes inflamed, the patho-gic process is typically osteomyelitic and as uch may advance to suppuration, abscess for-ation and sequestration. If air cells are pres-nt, the bone is spoken of as pneumatized. The ells contain a varying amount of air and are ined with a mucoepithelial membrane similar to hat of mastoid cells. If such a bone becomes nfected, the pathologic process becomes petro-itis, which is osteitis in contrast to osteomyelitis f the myeloid type. The pneumatized cells will e filled with acute inflammatory exudate, ulti-mately suppurating and forming pus. Abscesses occur, and sequestration of bone follows. Therefore, whether the petrosa consists of marrow or is pneumatized, the pathologic process differs only in the early phases; the terminal stages of suppuration are identical. However, the pneumatized bones are more liable to become infected.

Presented before the Section of Otolaryngology of he New York Academy of Medicine, Jan. 20, 1943.

CASE 1

J. D., a man aged 35 years, a patient of Dr. E. P. Fowler, of the Manhattan Eye, Ear and Throat Hospital, was admitted to the hospital on April 7, 1934, with the complaint of running right ear. The diagnosis was acute mastoiditis on the right side. His past history and the family history were irrevelant. Ten days before admission he had pain in the back of the head and behind the right ear. Three days later the ear began discharging, and five days after that the pain stopped. He noted inability to close the right eye. He did not remember the right side of the face being exposed to a draft within the past two weeks. He had no pain in the ear or the mastoid area and no headache at the time of examination. The heart and the lungs were normal. The reflexes were normal.

Simple primary mastoidectomy was performed April 7. The cortex was lifted with a gouge. Pus was found. The mastoid process was partially sclerotic. The cavity was cleaned out. The cells in the tip were broken down. They showed more involvement than was observed around the antrum. The mastoid tip was taken off and cells cleaned out. There were cells along the floor of the mastoid antrum which seemed to undermine the posterior semicircular canal, but these were left undisturbed. The dura above the tympanum was surgically exposed. The wound was packed with a sulfonamide compound in the form of powder and left open. A reaction to faradic current was obtained on the facial nerve but was slightly diminished from the normal.

Staphylococcus aureus and a hemolytic streptococcus were isolated from the infected area.

On April 8 the patient felt much better. There was some headache. The facial palsy was not improved. A response to faradic current was obtained. The patient could taste as well on the paralyzed side as on the other side, indicating that the lesion was below the point where the chorda tympani comes off—perhaps at the stylomastoid foramen, over which pus was under pressure and the cells were in particularly bad shape. Five days later spontaneous movement of the lower eyelid was better. There was slight movement of the lower lip. The wound was closing and beginning to fill from the bottom. Twelve days after operation the patient was discharged to the clinic.

One week after leaving the hospital he was readmitted. He had felt all right until two days before; then he began to have pain over the right temporal

region. He took his temperature at home and found it to be 104 F. He also had vomited two days before admission. The wound was dressed at the clinic that day. His eyes reacted normally. There was no acute involvement of the throat or of the left ear. The chest and the abdomen disclosed no abnormality. The reflexes were normal. The temperature was 103 to 104 F. A blood culture showed no growth after seventy-two hours' incubation. The patient was perspiring profusely and at times was chilly. His temperature in the next few days varied from 103 to 98 F., and he became symptom free. The hemoglobin content of the blood was 60 per cent. The red cell count was 4,000,000; the white cell count, 10,200; the differential count showed polymorphonuclears 81 per cent and immature cells 19 per cent. The urine had a specific gravity of 1.022. It contained no albumin or sugar. There was a faint trace of acetone and of diacetic acid. There was no indican. Eight days after this admission the patient was discharged.

Ten days later, on May 8, the patient was admitted for the third time. Since leaving the hospital he had returned for a dressing of the wound every two d... At the time of admission he was returning for pl... closure of the mastoid wound. The wound lo... fairly clean. The tampons were not detached. T... heart and the lungs, the eyes, the nose and the th... showed no changes from the findings of the previ... examination. The impression was that of an unhe... mastoidectomy wound. Plastic closure of the mast... wound was performed, and the patient was discha... May 10. On May 17 a roentgenologic report w...

Fig. 1 (case 1).—*A*, section through the normal left petrosa, reversed; *B*, section through the diseased ri... petrosa.

made as follows: Anteroposterior views show consi... erable lack of density of the tip of the right petro... bone, indicating bony absorption in the apex. T... cortex seems to be intact, but there is no evidence ... cell structure; cell structure is present on the le... Vertical views seem to confirm this appearance and al... indicate a pathologic process at the apex of the ri... pyramid.

On May 18 the patient was readmitted for the four... and last time. The diagnosis was meningitis, petrosi... and orbital abscess. Gradenigo's syndrome was prese... Radical mastoidectomy was performed on the right si...

exploration of the petrous tip. The cavity was filled granulations, which were curetted out. The bridge taken down and the horizontal semicircular canal wed into the antrum. The wall of the posterior l was taken down. There was an exposure of in the region of the zygoma. The entire tym-m, which was quite small, was opened. Bone was oved enlarging the original dural exposure until h of the middle fossa was exposed. An opening oft bone at the region of the eustachian tube was wed into the pyramid, until the dura was exposed. ttempt was then made to effect an entrance above horizontal canal. The little bridge of bone was oved, and with a blunt instrument the area around petrous tip was explored. Pus and fluid under eased pressure were found. More bone was removed eriorly and superiorly. Two drains were placed the cranial cavity and sewed to the skin. Much he lateral venous sinus was exposed, but it was not rkable pathologically. The eustachian tube was tted.

n May 18 the spinal fluid showed 50 cells—25 per polymorphonuclear leukocytes and 75 per cent small phocytes; on May 21 the corresponding counts were 26 and 74.

n May 21 a second exploration of the petrous tip made. The superficial granulations of the middle were cleaned away, and further curetting was mpted in the region of the tensor tympani muscle. bone here and about the eustachian tube was e soft. In curetting in the zygomatic region the er part of the glenoid cartilage was exposed. A t flat instrument was run beneath the dura and gasserian ganglion to the petrous tip. No epidural ess was uncovered. In curetting in the lower part he tympanic cavity a pocket of creamy yellow pus uncovered in the cells in the region of the jugular behind the wall of the posterior canal. A further mpression of the brain was necessary because of rupture of a branch of the posterior meningeal ry. The bleeding was controlled. The bone was oved over the lateral sinus. In the corner near utman's triangle a sharp spicule of bone punctured dura, causing cerebrospinal fluid to flow into the nd. Curetting behind and below the horizontal icircular canal, the surgeon suddenly uncovered a ket of pus which led through a fistula 1.5 cm. or more to the petrous tip. Another small pocket of pus found behind the sigmoid sinus.

he paralysis due to involvement of the sixth nerve red completely after the operation of May 18, but May 21 it reappeared and there was in addition ked edema of the eyelids on the right. There was headache, no stiff neck and no Babinski or Kernig , and the position sense was intact. The jaw, ch had been very stiff, was slightly less so. On y 23 the patient was vomiting and felt slightly y. There was no headache but the neck was tly stiff. The placing of the finger to the nose not as well executed as on the day before. There no nystagmus. The paralysis of the lateral rectus cle was less marked; in fact it was almost gone. spinal fluid contained 630 cells, 88 per cent of ch were polymorphonuclear leukocytes and 12 per small lymphocytes. On May 24 the patient looked h worse and continued to vomit. There was marked agmus on swinging the eyes to the right. The alysis of the lateral rectus muscle was almost gone. re was no headache. The patient's beginning men-ts of the previous day was full blown, and it ared that he might have labyrinthitis as well. Both ils were equal and reacted to light and in accommodation. There was weakness of the aforementioned right rectus, also of the left rectus medialis muscle. Both ocular disks looked fuzzy, particularly on the nasal side. Bilateral papilledema was present (about 0.75 diopter) and minute spots were noted which might have been hemorrhages but could not be clearly seen. The patient's temperature varied from 99 to 105 F. On May 25 the spinal fluid contained 2,500 cells—90 per cent polymorphonuclear leukocytes and 10 per cent small lymphocytes. Culture of the fluid showed the hemolytic streptococcus. On May 26 the patient died. Both petrous bones were removed and serial sections made. In figure 1 photographs of microscopic sections of both the normal and the diseased petrosa are shown.

CASE 2

G. N., a boy aged 9, a patient of Dr. J. R. Page, of the Manhattan Eye, Ear and Throat Hospital, was admitted on May 16, 1935, with the chief complaint of discharge from the left ear. Two days earlier he began to have earache on the left side, following an infection of the upper respiratory tract. The ear started to discharge and the discharge had continued. There was no pain. The past history revealed that he had had acute purulent otitis media on the left side one year before admission and on both sides in early childhood. His past history also included chickenpox, in April 1935, measles and pertussis. The family history was irrelevant.

The eyes were normal. The external canal of the right ear was normal. The drum membrane was white and somewhat thickened, with diminished luster; there was some congestion along the handle of the malleus. The left ear showed slight purulent drainage. The external canal showed no changes. The drum membrane was red, thick and bulging in the posterior half. There was a small perforation in the anterior lower quadrant. There was no tenderness over the mastoid process. The nose and the throat revealed nothing of importance. The tonsils were out. A few small glands were palpable in the left side of the neck. The red blood cell count was 4,970,000; the white cell count, 9,200, of which 64 per cent were polymorphonuclear leukocytes, 6 per cent large lymphocytes and 3 per cent small lymphocytes. Roentgen examination showed the left mastoid process to be of the pneumatic type, with 4 plus involvement and softening of the lower half.

Five days after admission simple mastoidectomy was performed on the left side. The bony cortex was opened. The mastoid process was soft and necrotic. The diseased bone was exenterated. The sinal and dural plates were cleaned. A small area of the cerebral dura of the middle fossa near the root of the zygoma was surgically exposed. The zygomatic cells were curetted. The antrum and the aditus were opened widely. The bony horizontal semicircular canal was exposed. A small part of the lateral sinus above the knee was surgically exposed. A large group of zygomatic cells was found. A group of infected cells was found under the sinus in the region of the jugular bulb and curetted. The edges of the dural exposure were enlarged. The cavity was irrigated with alcohol.

A culture of material taken at operation showed the hemolytic streptococcus.

On June 1, twelve days after operation, the boy did not appear acutely ill. He had no headache, dizziness or other complaint. The pupils were equal and reacted normally. The visual fields and the fundi were normal. He heard a tuning fork with the left ear. He had no stiff neck and no Kernig, Babinski or Oppenheim sign. There was no apparent involvement of the nervous

system. On June 9 a blood culture was negative when the temperature was 101.4 F. On June 11 the patient vomited several times. Since operation, twenty-one days earlier, he had complained of frontal headache on the left side off and on—at times it was quite severe. His disposition had changed somewhat in the past week; he was not as cheerful as before and often was drowsy. There were twitchings of both sides of the face. On June 11 a roentgenogram showed moderate absorption in the ethmoid cells. On July 19 the patient was discharged to the clinic to return later for plastic closure of his wound.

On August 20 he was readmitted to the hospital for secondary closure of the wound in the left mastoid region. The wound was wide open but filled w granulation tissue. The cutaneous margins were un mined and freed. The margins of the wound w freshened, and the wound was cleansed with 3 per iodine. On August 28 the wound was healing ti with a small amount of drainage. The patient discharged to the clinic.

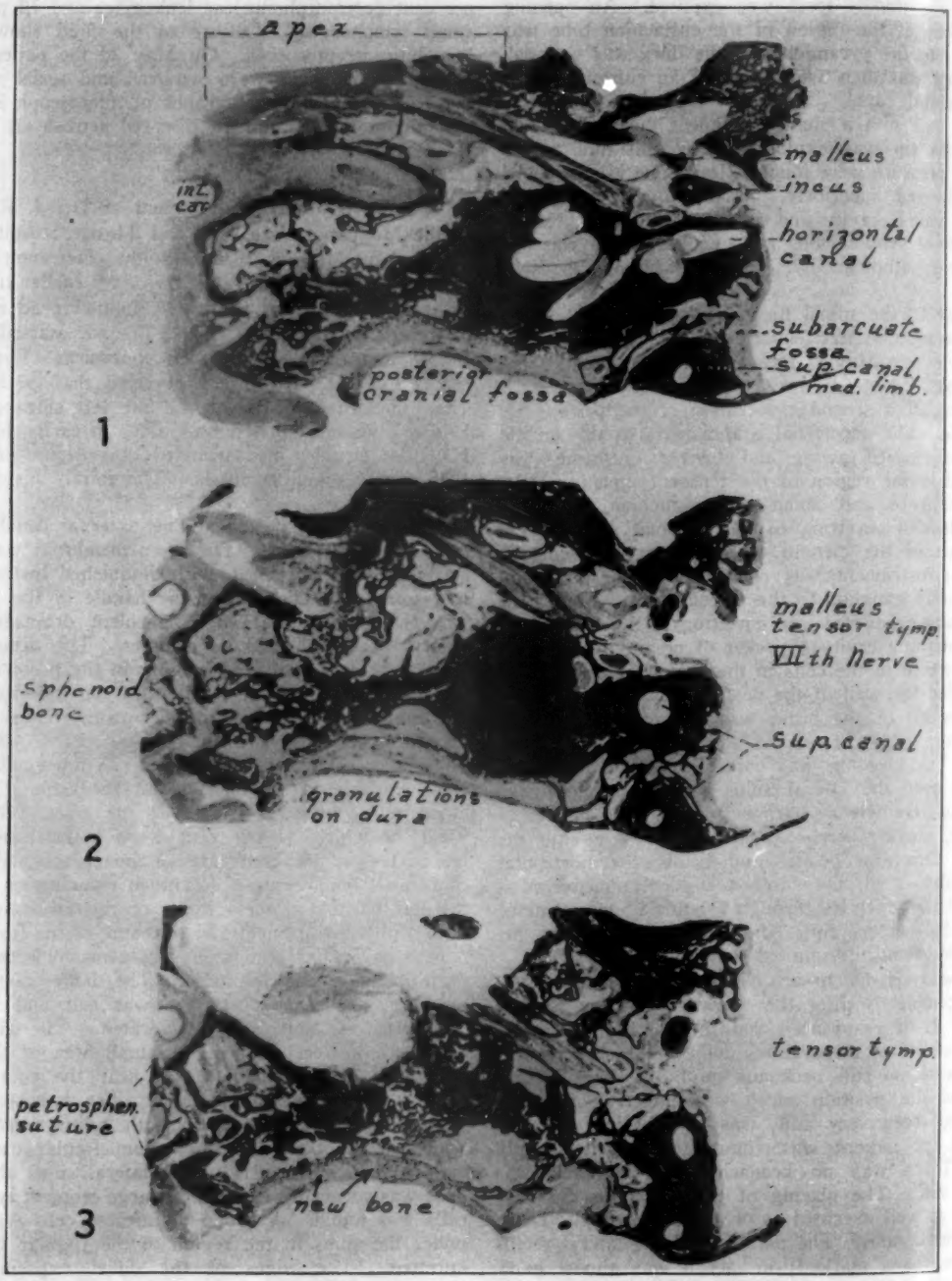

Fig. 2 (case 2).—Sections through the diseased right petrosa.

In the spring of the following year, April 21, 19 he was admitted for the third time. About five we earlier he had mumps. He had complained of p in the right and not in the left ear on and off for past few weeks. Six days before admission the ri ear began to drain purulent material. This draina persisted. The boy, now 10 years old, appeared w

rished and well developed. He was up and about did not appear acutely ill. From the right ear re was a copious discharge of greenish yellow mucoulent material. It was not possible to obtain a isfactory view of the drum membrane. There was swelling in the region of the mastoid antrum, but toidectomy on the left side and a pneumatic type of right mastoid process with severe involvement (4 plus) and marked softening. Culture of material obtained by myringotomy on the right side showed the same organism as was found a year earlier—the hemolytic streptococcus.

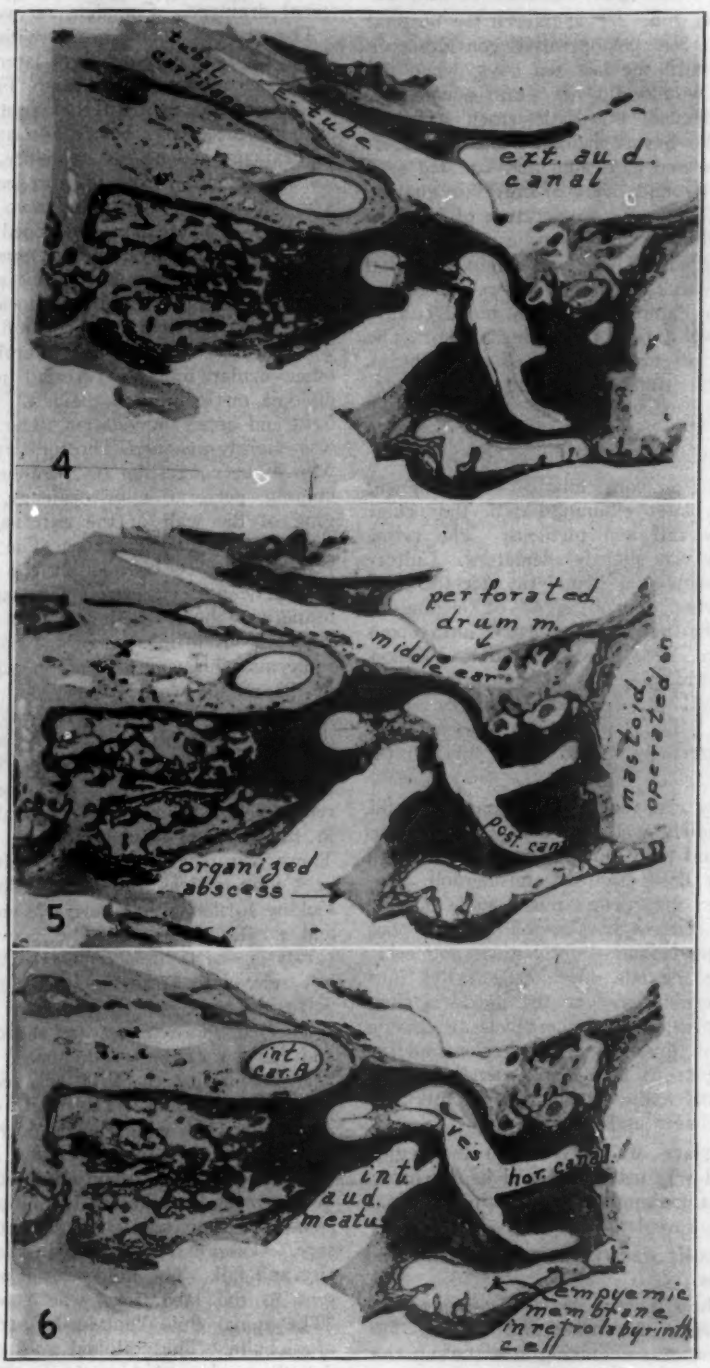

Fig. 3 (case 2).—Continuation of sections through the diseased right petrosa.

e tip of the mastoid process was quite tender to essure. The left ear showed no pathologic condition. here was slight rhinitis. The throat was normal. he glandular system was normal. The observations the chest, including the heart, were not remarkable. entgenograms showed the expected evidence of mas-

On April 27 simple mastoidectomy was performed on the right side. The pneumatic cavity in the mastoid process was very hemorrhagic. Some foul pus was present. Culture of the pus yielded the hemolytic streptococcus. The lateral sinus was surgically exposed at the knee over an area 3/8 by 1/8 inch (1 by 0.3 cm.).

Two surgical exposures of dura in the middle cranial fossa were made. Tip cells, post angle cells and zygomatic cells were removed. The wound was cleansed with surgical solution of merbromin N. F.

The patient's temperature was 100 F. at the third admission, and it fluctuated even after operation from 97.2 to 99.4. He was discharged improved on May 12.

Ten weeks later, on June 27, he entered the hospital for the fourth time. His postoperative convalescence had been uneventful until the last ten days, when the superior part of the wound did not drain adequately. The wound was reopened, but satisfactory drainage could not be obtained. Severe frontal headache developed. The patient became nauseated and vomited several times. The heart sounds were of only fair quality. The rate was slow in view of a temperature of 102.5 F. There was a soft medium-pitched systolic murmur over the base, not transmitted. There was slight pulsation of the vessels in the neck. The plantar response was poor. There was no Babinski sign. A blood culture was negative after twenty-four hours' incubation. The patient was carefully observed for abscess of the brain, meningitis and lateral sinus thrombosis. Roentgenograms made on June 29 showed marked erosion on the superior ridge from the internal meatus to the apex of the petrous pyramid.

On July 2 the right temporal lobe of the brain was explored. Five days later examination of the ocular fundi disclosed veins full and tortuous. The retina surrounding each disk was slightly edematous. Culture of the spinal fluid showed a hemolytic streptococcus. On July 9 there were clinical signs of meningitis. Beginning July 8 the patient had daily intravenous injections of 1,000 cc. of 5 per cent saline solution, followed by spinal drainage, as well as many blood transfusions. On July 17 the spinal fluid did not appear to be under pressure. It was possible to draw off only 5 cc., which was extremely turbid. On July 20 the patient died.

Autopsy.—The body was that of a 10 year old child, well developed and fairly well nourished. There was a mastoidectomy wound with dura exposed on the right side. Removal of the skull disclosed considerable serofibrinous exudate over the cortex, much more marked over the base, in the cisterna basalis and over the pons and the medulla oblongata and slightly more noticeable on the right than on the left. Removal of the dura disclosed a hemorrhagic area over the apical portion of the right petrous pyramid, and there was an erosion of bone, 2 cm. by 1 cm., involving the posterior superior border of the right petrous bone and extending down and around the internal auditory meatus. In this area the bone was hemorrhagic and eroded.

On section the surface of the brain in the region of the mastoid wound was macerated and hemorrhagic, probably the result of manipulation. Multiple sections of the brain failed to reveal any abscess.

The anatomic diagnosis was right-sided petrositis and suppurative meningitis.

Figures 2 and 3 show several serial sections of the petrosa and of the area of erosion and ulcers. The sections are longitudinal ones beginning near the superior surface of the petrosa and extending downward.

CASE 3

G. T., a white boy aged 9, a patient of Dr. M. T. Smith, of Grasslands Hospital, Westchester County, N. Y., was admitted to that hospital on May 11, 1937 with a chief complaint of double vision when looking to the left. The family history was irrelevant. There had been a previous stay in the hospital from Feb. 28 to March 5, 1935. The diagnosis at that time was bilateral acute non purative otitis media. There had been drainage the left ear and pain in the left eye with tra paralysis of the left lateral rectus muscle. Meni developed—an infection with type 1 pneumoco probably secondary to petrositis. The patient treated with type 1 pneumococcus serum and f spinal drainage. Complete recovery followed.

The present illness began with bilateral earach April 18, 1937. Three days later the family phy did paracentesis on both ears. Following this, the ear drained more than the right. Both ears st draining three days before admission. There had no drainage and no earache since. On April 24 s pains developed across the forehead and in the left The patient was unable to move his left eye later On May 8 severe pain developed in the right eye, there was no paralysis in the muscles of this eye. patient vomited twice during the week before admis

On admission the patient appeared to be well de oped, fairly well nourished and comfortable. T was paralysis of the left lateral rectus muscle. other ocular muscles revealed paralysis. There diplopia on looking to the left. The pupils reacte light and in accommodation. The right drum memb was slightly opaque. The light reflex was lost. T was slight congestion of Shrapnell's membrane bu bulging, perforation or drainage. There was no ging of the wall of the external canal or tender over the mastoid process. The left drum memb was also slightly opaque, but the light reflex was p ent. The posterosuperior quadrant of the drum m brane was slightly yellow with a suggestion of bul The mucous membrane of the nose was congested, some mucopus was present on each side. The find in the mouth were not relevant. There was no dence of inflammation of the throat. The tonsils out. A slight postnasal drip was evident. The showed no rigidity and no marked adenopathy. findings in the thorax and the abdomen were remarkable. Neurologic examination revealed para in the area of distribution of the left sixth n There was pain over the left fifth nerve. No ch in sensation was observed. There was diplopia looking to the left. There was no stiffness of the and no Kernig sign. The provisional diagnosis paralysis due to involvement of the sixth nerve on left side, with Gradenigo's syndrome, due possibly petrositis.

The patient's temperature on admission was 100.2 and for three days it fluctuated between 99 and The blood showed 4,100,000 red cells, with nor hemoglobin content. The white cell count was 13, 79 per cent of which were polymorphonuclear le cytes. The urine was normal. A culture of mat taken from the throat on admission was negative. Wassermann reaction of the blood was negative. spinal fluid on admission seemed under normal p sure. Compression of each jugular vein gave a nor rise and fall. The fluid was clear. A 15 cc. speci sent to the laboratory was reported on as follo "The spinal fluid contained 6 cells and a faint t of globulin. The colloidal gold curve was 1222100 A culture showed no growth after twenty-four ho The sugar content was 69 mg. and the chloride con 709 mg. per hundred cubic centimeters." It is inter ing to note that the patient had no complaints for first forty-eight hours in the hospital, but on May he complained of pain in the right ear, the right of the forehead and the right eye during the ni Examination of the eyes, the ears, the nose and throat revealed nothing new. On this day the pat

s seen by Dr. Smith, who evaluated the case as lows: "The left drum shows slight thickening and lack of luster in the posterior half, but there is no finite bulging and no sign of an acute inflammatory ocess. The right drum is intact, with landmarks sible, but is somewhat gray and lacking in luster; re are no signs of an acute inflammatory process present. The hearing is roughly acute in each ear, there are no clinical signs of mastoid involvement. amination of the nose and the throat gives essentiy negative results. There is complete paralysis in area of distribution of the left sixth nerve. No er signs of involvement of cranial nerves can be cited. The patient is alert and has no complaints present. His general condition is satisfactory. The pression is that of resolving bilateral otitis media

The boy complained of pain in the head and the back. The left drum membrane was incised at this time, with evacuation of a slight amount of serous material; a twenty-four hour culture of this showed staphylococci, probably from contamination. A spinal tap showed normal pressure of the spinal fluid. The fluid was definitely cloudy, and cells were too numerous to count. Culture of the fluid showed type III pneumococcus.

Simple mastoidectomy was performed on the left side. Several small cortical perforations were found in the middle of the exposed mastoid process. However, no pus exuded from these perforations. When the cortex of the mastoid process was removed, no pus was seen in the more superficial air cells. However, as these were curetted away, isolated groups of purulent cells were found posteriorly close to the inner

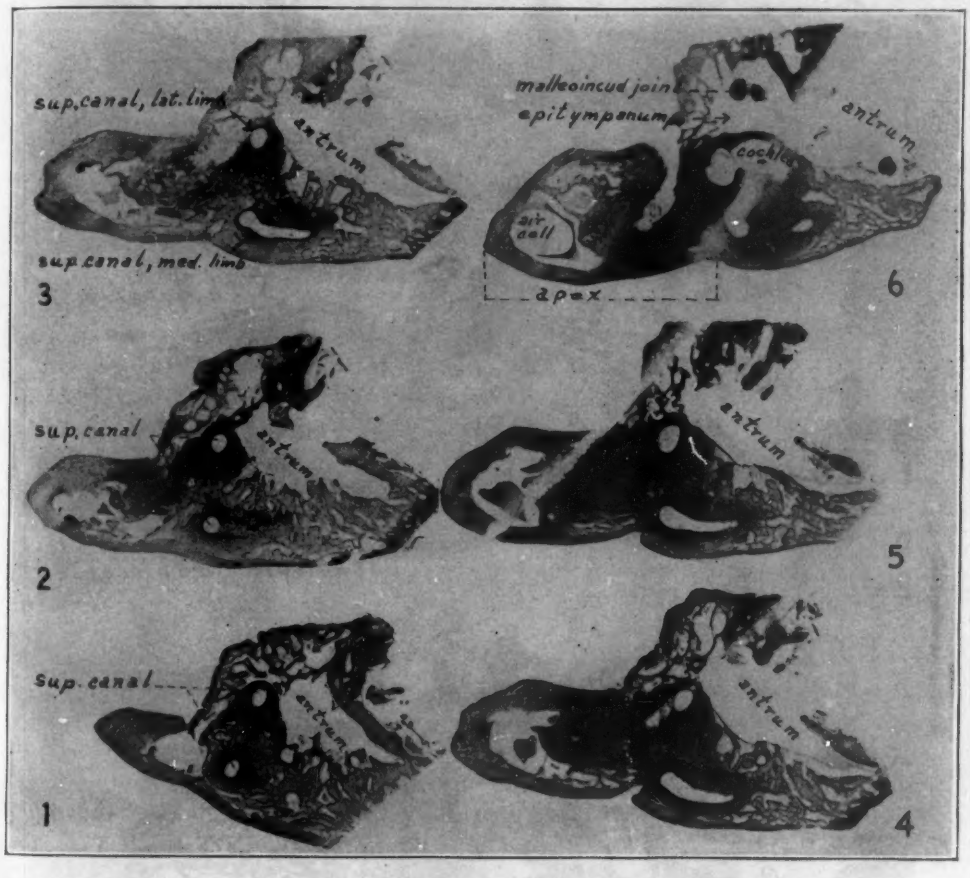

Fig. 4 (case 3).—Sections through the normal right petrosa.

hout signs of mastoid involvement and without clini- signs except those relating to the sixth nerve to gest petrositis."

A roentgenogram of the chest showed lymphoid ulation in the hilar region, probably tuberculous. ntgenograms of the paranasal sinuses revealed ing remarkable. The Mantoux test showed a posie reaction with a 1:1,000 dilution of tuberculin. rther cultures of the spinal fluid were negative. e patient's course was uneventful until May 17 (six after admission), when he vomited several times. e vomiting was nonprojectile. The patient was carey examined and showed nothing additional except reased drowsiness. The temperature was 100.6 F. early evening definite signs of meningeal irritation developed. The temperature had risen to 103.8 F. udzinski's signs and Kernig's sign were positive.

table. There was no breaking down. However, a moderate amount of purulent material came away from the antrum and antral regions when these were exposed. The sinal plate was normal. A piece of the plate was then removed and the sinal dura appeared normal. There was a small operative exposure of the dura anteriorly. There was some pneumatization found posterior to the posterior semicircular canal; the cells here contained some soft material, somewhat purulent. No fistula into the petrous portion, however, was discovered. The wound was washed with saline solution and lightly packed with iodoform gauze.

On the following morning the patient's condition was unimproved. He was delirious, and Brudzinski's and Kernig's signs were more markedly positive, with increasing rigidity of the neck. The temperature was 103.2 F. A diagnosis of generalized septic meningitis

was made. Because the patient was successfully treated previously by use of serum and forced spinal drainage, this was repeated, and in addition 58 cc. of azosulfamide [1] was administered in the first twenty-four

order to furnish surgical drainage with a view relieving the meningitis.

On the same day radical mastoidectomy was do on the left side. The wall of the posterior canal a

Fig. 5 (case 3).—Sections through the diseased left petrosa.

hours. Then another attempt was made to find a fistulous tract leading into the petrous pyramid in

the bridge were taken down; the ossicles were remove and the middle ear cleaned out. Nothing unusual wa found in the middle ear. There was no evidence o pus or granulations. The patient had a large eusta chian tube. The epitympanic space and the regio above the tube were carefully searched for possibl

1. Azosulfamide is disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonate.

rosis leading toward the petrous tip, but none was nd. Removal of the tegmen antrae, the tegmen ympani and a portion of the squamous portion of temporal bone disclosed that the dura and dural ssels were injected and palpation showed that there s tension. Multiple slits were made in the dura the under surface of the temporosphenoid lobe and in that on the lateral surface. No cerebrospinal d escaped, but what appeared to be thick pus or ebral tissue promptly presented in the incisions.

The patient left the operating table in poor condi-n. He was in poor condition when seen in the rd. Some bleeding was observed, and a postnasal g was put in and the nares packed. The bleeding s controlled. The patient became rapidly worse and d three hours and a half after operation. Permis-n for postmortem examination of the head was ained.

Autopsy.—Examination of the head showed extreme ilar meningitis. Grossly, little definite pathologic eration was evident in the petrous bones. This is quently true.

COMMENT

There are at least four pathways of extension an infection of the middle ear or the mastoid trum to the petrosa: the retrolabyrinthine ute, the supralabyrinthine route, the anterior byrinthine or pericarotid route and the infra-byrinthine or hypotympanic route. The vari-s pathologic pictures in petrositis may be due the different routes of extension of the infec-n. Likewise the virulence of the organism and e resistance of the patient as well as the type chemotherapy may determine the character of thologic changes present. Extension of the fection intracranially in the form of meningitis of abscess of the brain may be by retrograde lebitis or by direct extension through continuity or contiguity of the tissues. If an abscess is present in the petrous pyramid, it may rupture into the meninges or more rarely through the basilar plate of the petrous bone into the tissues of the neck and eventually into the nasopharynx. These 3 cases clearly demonstrate different petrosal locations of abscesses, of which there are many others. Each location calls for a different surgical approach. Clinically the disease in each of the 3 cases ran a prolonged and recrudescent course, requiring repeated hospitalization of the patient. Every case of prolonged mastoiditis or relapsing meningitis should be carefully surveyed with petrositis in mind. It is frequently difficult or impossible to locate the infected area either at surgical exploration or at autopsy. This is particularly true if the dura is intact. Serial microscopic sections of the bone are necessary to find and properly evaluate a petrosal infection. Proper surgical drainage of all original foci of infection is essential to cure. The surgical procedure must be individualized as to the case and the surgeon. With proper surgical management combined with early administration of heroic doses of a sulfonamide compound there is a fair chance of survival of the patient. Heroic doses should be employed because the mortality of the disease greatly overbalances the toxicity of the sulfonamide drugs. Further the large doses should be continued from three to four weeks after the patient is clinically well. This is necessary to avoid recrudescences of the infection. If relapse occurs, the infection may not respond to renewed doses of the drug.

653 Park Avenue.

Case Reports

PAIN REFERRED TO THE FACE, NECK, UPPER EXTREMITY AND CHEST DUE TO LESIONS IN THE EAR

Report of Two Cases

HENRY P. SCHUGT, M.D., NEW YORK

Pain due to pathologic conditions in the ear simultaneously involving such large areas as the face, the neck, the arm and the chest is an uncommon occurrence. The 2 patients described here were treated for chronic mastoiditis. An explanation of the neurologic pathways according to present knowledge will be given.

REPORT OF CASES

CASE 1.—Mrs. L. V. underwent radical mastoidectomy on the right side in 1932. A cholesteatoma was found in the mastoid antrum and middle ear with extensive destruction of bone, including the facial canal. Postoperative facial paresis persists up to the present time. The mastoid wound healed and stayed dry until July 1942; then it began to discharge again after an infection of the upper respiratory tract. Severe pain developed in the ear, and the patient was treated until November 1942, when she was readmitted to the hospital for observation. Repeated general examinations by the medical department showed no involvement of the lungs, the heart or other internal organs. The eyegrounds and the neurologic findings were within the normal range. Culture of material from the ear gave a growth of Staphylococcus aureus. A revision of the mastoid cavity showed a small cholesteatoma near the opening of the eustachian tube. It was during the after-treatments that pain became extremely severe, especially during cleansing and after packing of the mastoid cavity. It was noted that the pain started when the anterior wall of the mastoid cavity or when the eustachian tube was touched or packed with a thin one-quarter inch (0.64 cm.) tampon. The pain would instantly spread over the entire auricle, to the face, the eye, the temple, the upper and the lower lip, the neck, the upper arm and into the chest (fig. 1); it would last all day and most of the night and yield only to strong sedatives and local applications of a 10 per cent solution of cocaine. There was marked pulsation in the eustachian tube, which during the periods of pain would extend along the cervical tissues, especially in the region of the large vessels. There was pronounced tenderness of these tissues, with distention of the external jugular vein. Examination of the neck showed no enlarged cervical glands and no signs of inflammation along the large vessels. It was decided to revise the mastoid cavity again. A thorough inspection and curettage of the area of the eustachian tube was done without any further pathologic process being found. The pain, however, recurred in the same manner and with the same intensity as after the first revision. Subsequently a perinephritic abscess developed, which was operated on. The mastoid cavity was now [illegible] unpacked, and gradually complete epithelization [illegible] place, and the pain finally subsided. The patient [illegible] discharged from the hospital with both the mast[illegible] cavity and the perinephritic abscess healed.

CASE 2.—Mrs. W. had chronic otitis media on [illegible] left side and was admitted for observation at about [illegible] same time as the patient in case 1. During this ti[illegible] severe pain developed in the ear as well as in the l[illegible] side of the chest and in the upper arm, and the pati[illegible] complained of pain in the region of the heart. T[illegible] pain was less violent than in our first patient and w[illegible] felt deep in the chest, not below the fourth rib, howev[illegible] No local tenderness to pressure could be made out eit[illegible] in the thorax or the upper arm. A thorough examin[illegible] tion of the chest and the heart, including electrocardi[illegible] graphic investigation, revealed no pathologic conditi[illegible] A modified radical mastoidectomy was performed, wh[illegible] had to be revised, with subsequent healing of the mast[illegible] wound and preservation of hearing. The pain in t[illegible] chest and the upper arm subsided after the first oper[illegible] tion and was not noticed during after-treatments as [illegible] case 1. There was no radiation of pain into the fa[illegible] or the neck at any time.

No doubt the pain sensations described f[illegible] under the classification of referred pain[illegible] According to Head, referred pains are foun[illegible] within segments of the body that are supplied b[illegible] nerves which also supply afferent fibers to th[illegible] site of the lesion. Assuming that the site [illegible] the lesion in the first case was within the distr[illegible] bution of the trigeminal nerve, the pain exper[illegible] enced in the temple and the face may be co[illegible] sidered as a referred one conforming with Head[illegible] theory. The same can be said about the pain [illegible] the upper cervical region, since the trigemin[illegible] spinal tract reaches the level of the upper cervic[illegible] nerves. Pain sensation in the arm and th[illegible] thorax due to lesions in the ear and the nas[illegible] cavities has been investigated by Kuntz[illegible]

1. Kuntz, A.: (*a*) Pathways Involved in Pains [illegible] Nasal and Paranasal Origin Referred to the Low[illegible] Cervical and Upper Thoracic Segments and the Upp[illegible] Extremity, Ann. Otol., Rhin. & Laryng. **45**:394 (Jun[illegible] 1936; (*b*) Nerve Fibers of Spinal and Vagus Orig[illegible] Associated with the Cephalic Sympathetic Nerves, ib[illegible] **43**:50 (March) 1934; (*c*) The Autonomic Nerv[illegible] System in Relation to Otolaryngology, J. A. M. A. **10**[illegible] 334 (Aug. 1) 1936.

ccording to his findings and to clinical obser-ations by Foerster,[2] Temple-Fay [3] and others, fferent branches of the upper thoracic nerves xtend along the carotid arteries and afferent bers of vagus origin join the plexuses on the nternal and external carotid arteries and reach

Fig. 1.—Pain referred to a large area, due to lesions the ear.

e area of the trigeminal nerve. Afferent im-ulses therefore arising in primary lesions such s those in the mastoid area in our cases are con-ucted from the site of the lesion into the spinal ord through afferent components of the thoracic erves, which traverse the nerves associated with e common, internal and external carotid rteries. The autonomic components of referred ains localized in the lower cervical and upper horacic segments and the upper extremity which e due to lesions in the mastoid area can thus be explained as in the following quotation from Kuntz,[1c] illustrated by figure 2:

> . . . Impulses arising at the site of the lesion reach the upper thoracic segments of the spinal cord through afferent spinal nerve components which traverse the plexuses on the common and internal carotid arteries. These fibers effect connections with visceral efferent neurons in the spinal cord, through which the impulses are conducted into the corresponding ganglions of the sympathetic trunk, where they are transmitted to sympathetic neurons, through which they are conducted toward the periphery. The impulses that arise in the peripheral pain receptors are conducted into the spinal cord through afferent components of the corresponding spinal nerves and upward in the lateral spinothalamic tract on the contralateral side.

This explanation is based on Werne's [4] experimental work concerning the mechanism of referred pain, namely, that referred pain associated with visceral lesions has its origin in changes in the skin and the underlying tissues through vis-

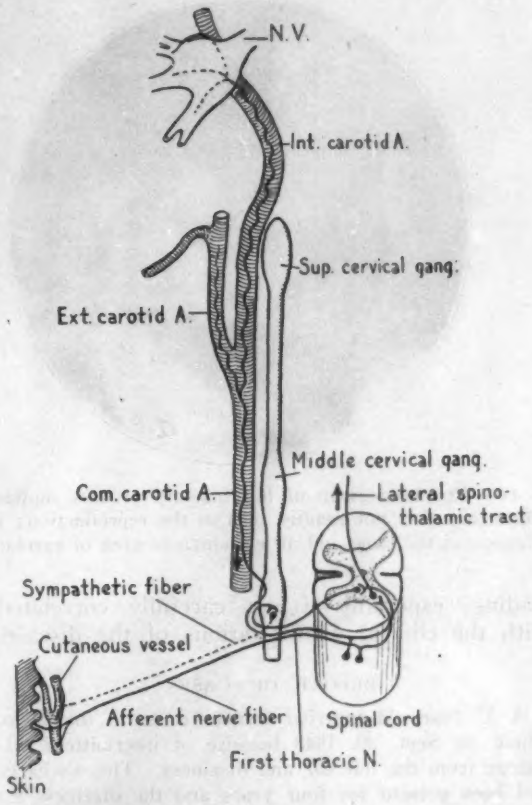

Fig. 2.—Pathways of pain referred to the neck, the thorax and the upper extremity (after Kuntz, modified).

cerocutaneous reflexes; in other words, the sensation of pain is the direct result of afferent impulses which arise in the periphery.

122 East Seventy-Sixth Street.

2. Foerster, O.: Die Leitungsbahnen des Schmerzgefühls, Berlin, Urban & Schwarzenberg, 1927. Foerster, O.; Altenburger, H., and Kroll, F. W.: Ueber die Beziehungen des vegetativen Nervensystems zur Sensibilität, Ztschr. f. d. ges. Neurol. u. Psychiat. **121**: 139, 1929.

3. Fay, T.: Atypical Facial Neuralgia: A Syndrome of Vascular Pain, Ann. Otol., Rhin. & Laryng. **41**:1030 (Dec.) 1932.

4. Werne, T. B.: The Diagnostics of Pain, Copenhagen, Levin & Munksgaard, 1936.

CHOLESTEATOMA WITH FISTULA INTO THE LABYRINTH

Report of a Case in Which the Roentgenologic Findings Were Confusing

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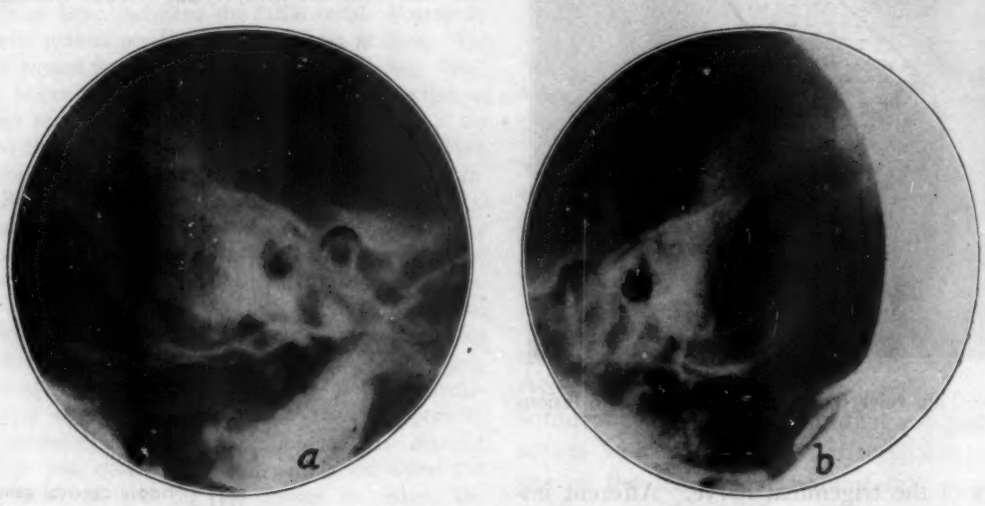
In the case to be reported, cholesteatoma with fistula into the labyrinth on the left side was diagnosed clinically, and the diagnosis was proved correct at operation. Roentgenographic examination of the mastoid processes, however, revealed indefinite, if any, evidence of disease on the left side but marked evidence of abnormality on the right side. The case is reported to illustrate that roentgenographic evidence of chronic disease of the ear may at times be misleading, especially if not carefully correlated with the clinical manifestations of the disease.



(*a*) Roentgenogram of left mastoid process showing no evidence of abnormality; the dehiscence in the left epitympanum is not readily seen in the reproduction; (*b*) roentgenogram of the right mastoid process, showing sclerosis of the bone and an epitympanic area of rarefaction, which was interpreted as a cholesteatoma.

REPORT OF CASE

A 37 year old married woman came to the Mayo Clinic on Sept. 30, 1943 because of intermittent discharge from the left ear and dizziness. The discharge had been present for four years and the dizziness for fourteen months. She first noticed a discharge from her left ear following a severe cold. At that time her family physician told her that the left ear drum was almost completely destroyed and that he suspected that she had had an infection of the ear in infancy or childhood. The discharge from the ear continued thereafter for several months until the administration of alcohol drops resulted in a cessation of the discharge. In the next two years she had several mild recurrences of the discharge but it never lasted more than a few days at a time.

Eighteen months before she came to the clinic the left ear started to drain again. The discharge was yellow but not profuse or foul smelling. After that recurrence the discharge continued for a whole year in spite of intensive treatment. Finally, the patient became so discouraged that she discontinued all treatment. The discharge stopped shortly thereafter. It seems probable that the continuous treatment acted as an irritant which was responsible for the continuation of the discharge.

Regarding her dizziness, the patient explained that the first attacks occurred about fifteen months before she came to the clinic. At that time she had three different attacks of rotatory vertigo, each of which lasted about two hours. Several weeks later she noticed an entirely different kind of dizziness, which she had from then on. She described this sensation as a lateral to and fro movement of all objects. The amplitude of these movements was in direct proportion to the distance of the objects from her, and their rate was synchronized with her heart beat. This dizziness was present practically all the time; it was worse when she was tired or laboring under some stress, and on those occasions it was accompanied by nausea.

About seven months before she came to the clinic she experienced a third type of vertigo. The attack was

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vere and rotatory in character; it awakened her in e middle of the night, was accompanied by nausea and omiting and forced her to spend the next five days in ed. (Complete perforation of the bony labyrinth may ccount for this sudden exaggeration of the symptoms f labyrinthine irritation.)

Examination of the left ear revealed a dry ear with a edium-sized attic fistula. The drum membrane was overed with hard crusts and could not be fully visualzed. The right drum membrane was thickened and omewhat retracted (grade 2 on the basis of 1 to 4) ut otherwise was normal. Tuning fork tests and an udiogram revealed moderate conduction deafness on the eft side. There was spontaneous nystagmus on looking o both sides, and the fistula test was mildly positive on he left side.

After the left ear had been cleaned, the tympanic membrane was found intact but markedly retracted (grade 3). During this cleansing process and during careful irrigation it was noticed that the slightest manipulation produced considerable discomfort and violent dizziness. Moreover, on one occasion cholesteatomatous material was removed from the fistula with an ear hook.

General and neurologic examination revealed no abnormalities. Roentgenographic examination of the mastoid regions revealed no abnormality on the left side except that the cells were small. The right mastoid process, however, was reported as being markedly sclerotic with an irregular, not well defined area of rarefaction in the epitympanic region, which was interpreted roentgenologically as a cholesteatoma (figure). The roentgenographic examination was repeated to make certain that the two sides were properly identified on the roentgenograms.

Knowing all of the clinical aspects of the case, we noted a dehiscence in the left epitympanum, which we thought might represent a cholesteatoma with fistula in the horizontal semicircular canal (figure). The dehiscence was not sufficiently well defined or sharply enough demarcated to make a purely objective diagnosis of this kind possible.

A Bondy modification of radical mastoidectomy, which was carried out by the endaural approach, verified the preoperative diagnosis. A large cholesteatoma was found in the left epitympanum. It had eroded into the anterior part of the horizontal semicircular canal and produced a large fistula. The cholesteatoma was removed, but the fistula was not disturbed. The Lempert incision was closed by a plastic procedure. The patient made an uneventful recovery, and the vertigo disappeared completely within ten days.

COMMENT

In this case of cholesteatoma with fistula into the labyrinth, the roentgenologic evidence indicated disease in the right mastoid process whereas the patient's difficulty proved to be on the other side. This case illustrates that a laboratory test, such as a roentgenographic examination, may be misleading, and that when laboratory findings conflict with clinical findings the greater weight should be given to the latter. We feel that the roentgenologic findings on the right side represent an ancient quiescent lesion.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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In this review of the literature we have aimed to present in a readable and practical way the useful and significant new knowledge that has been added to the field of progressive deafness. We have attempted to evaluate the contributions in editorial comments, enclosed in parentheses.

The literature for 1942 reflects not only the war but the growing importance of aviation in warfare; there are numerous articles dealing with the effects of aviation on the ear. The interest in avitaminosis as a cause of deafness shows a decline, if we may judge from the decreased number of articles dealing with this subject, while the growing interest in Ménière's disease continues to be evident. The surgical treatment of otosclerosis still receives considerable attention in the American literature, with but a single article from abroad.

The material is covered in the following order:

Otosclerosis
- Pathology and Etiology
- Medical Treatment of Otosclerosis
- Surgical Treatment of Otosclerosis

Chronic Adhesive Deafness

Aviation and Submarine Deafness

Primary Nerve Deafness
- Nerve Deafness from Acoustic Trauma
- Allergic Nerve Deafness
- Congenital Nerve Deafness
- Nerve Deafness of Unknown Cause
- Treatment of Nerve Deafness
- Nerve Deafness of Central Origin
- Ménière's Disease.

OTOSCLEROSIS

Pathology and Etiology.—Wittmaack[1] presents the clinical history and the histologic observations in an interesting case of otosclerosis. The patient was a prominent professor of anatomy who had been examined by many of the leading otologists, including Wittmaack, and who willed his temporal bones to the latter for study.

As a child the patient had been taken to von Tröltsch for inflations on account of the after-effects of scarlet fever.

Impaired hearing in the right ear was first noticed when he was in medical school, when the patient found that he could auscult better with his left ear.

At the age of 36 he consulted Schwartze (Halle, Germany), who found "a conduction deafness in the right ear; drum membranes not involved; therefore, probably of the stapes." Application of silver nitrate to the pharyngeal bands every few weeks and gargling of salt water were advised.

When the patient was 38, Mackentun (Leipzig, Germany) carried out frequent inflations without benefit. A year later the patient first noticed diminished hearing in his left ear.

At the age of 44 a pulsating roaring began in the left ear. An examination by Heine (Munich, Germany) resulted for the first time in the diagnosis of "sclerosis, hereditary, incurable."

When the patient was 46, Siebenmann (Basel, Switzerland) confirmed the diagnosis of otosclerosis but found the nerve of hearing in the left ear intact, and made a relatively favorable prognosis.

Wittmaack first saw the patient when the latter was 55 and found the drum membranes essentially normal; he observed a bilateral conduction type of deafness, greater in the left ear. Roentgenograms of the right and the left mastoid process showed complete absence of pneumatization of the right and marked limitation of pneumatization of the left. The differential diagnosis lay between deafness due to otosclerosis and deafness due to chronic middle ear adhesions. The marked limitation of pneumatization indicated recurring exacerbation of latent otitis

1. Wittmaack, K.: A Remarkable Case of Otosclerosis, Hals-, Nasen- u. Ohrenarzt (Teil 1) **32**:1, 1941.

ancy, which could have been the basis of an hesive process. However, the irresistible prog- s of the hearing defect and the pulsating nitus, which the author recognizes as of great gnostic significance for otosclerosis, indicated t otosclerosis was superimposed on the in- tile otitis; so the diagnosis of otosclerosis de by Heine and Siebenmann was concurred by Wittmaack.

At the age of 74 the patient died of coronary ease. There were no relatives, ancestors or scendants with deafness.

Histologic examination of the temporal bones vealed practically identical pathologic changes the two ears, as follows:

The drum membranes were thickened, chiefly ing to a hyperplastic fibrous increase in the ickness of the mucous membrane layer. This perplastic fibrous mucous membrane lined the ire middle ear cavity, with bands of fibrous sue across the niches of both windows enclos- g cystlike cavities filled with precipitated al- min and detritus.

The labyrinthine capsules showed typical oto- erotic foci as follows: A focus above and in nt of the oval window reached and almost rrounded the window. The otosclerotic bone the focus surrounded a large, dilated vein ich was a branch of the vein accompanying e nerve of Jacobson. Close to the vein the osclerotic bone showed marked deposition of ne, while in the periphery of the focus the st active stage of the otosclerotic process was n, with strikingly dilated varicose veins with- areas of resorption, the surrounding bone ining bright blue and filled with many bone ces and many bone cells. On the margins re finger-like projections of otosclerotic bone tending into the surrounding bone in the form widened vessel spaces, with marked blue stain- g of the the surrounding bone. A second vein m the venous network around the facial nerve tered the focus. The latter finding explains, cording to Wittmaack, the failure of his oper- on to arrest the process in some cases, for vation of the dura from the superior surface the pyramid would avulse the veins in the lian canal but would not affect those from the ial canal.

A focus in the stapes involved the anterior crus d the footplate and extended across the sta- diovestibular joint anteriorly to coalesce with focus anterior to the oval window. A mark- y dilated vein entered this focus also.

A third and a fourth focus were found in the sule of the cochlea, consisting of inactive oto- erotic bone with complete reossification of the as of resorption that represent the active stage. Several dilated veins entered each of these cochlear foci, partly from the veins that accompany Jacobson's nerve and partly from the vein that accompanies the superficial petrosal nerve. The latter vein entered a plexus on the roof of the petrous apex and extended forward to surround the meningeal artery close to the latter's departure from the skull. None of the veins from any of the foci communicated with the venous plexus around the carotid artery.

The membranous labyrinth showed evidence of a long-standing hypertonia with a recent slight hypotonic collapse of Reissner's membrane.

Wittmaack interprets the changes in the mucosa of the middle ear and the restricted pneumatization as the results of an exhausted latent infantile otitis of severe degree.

The presence of a large dilated vein within each otosclerotic focus Wittmaack interprets as evidence supporting his theory of the genesis of the disease: that it is the result of venous stasis or of a retrograde venous blood flow. Foci in the capsule of the cochlea had previously been thought by the author to be due to retrograde venous flow from the carotid plexus and therefore not amenable to his operation. As a result of his study of this case in which the cochlear foci were entered by veins from the surface of the pyramid, Wittmaack has now extended his operative indications to include primary nerve deafness diagnosed as due to cochlear foci of otosclerosis because the course is the typical course of otosclerosis with pulsating tinnitus. The latter symptom, to which the author attaches great significance, is explained as the pulsations of the meningeal artery transmitted through the retrograde venous blood flow. In several cases of primary nerve deafness diagnosed as cochlear otosclerosis in which the dura was surgically elevated from the superior surface of the pyramid, relief from or lessening of long-standing pulsating tinnitus already attests the favorable influence of the operation.

In conclusion Wittmaack points out that this case proves that the clinical diagnostic methods at one's disposal are adequate to diagnose during life the pathologic changes resulting from infantile otitis, as well as the presence of otosclerosis with ankylosis of the stapes. (Comment: We agree with Wittmaack that otosclerosis with stapedial ankylosis can be correctly diagnosed during life, the slow but irresistible progress of the impairment of conduction in the absence of acute inflammatory episodes in the middle ear characterizing the condition—for, as Wittmaack says, there is no other known process that can cause such a picture. With Wittmaack's statement that he can diagnose cochlear otosclerosis in

the absence of fixation of the stapes we would hesitate to agree, for to our knowledge such cases diagnosed correctly during life and confirmed by histologic study after death have never been reported. The usefulness of Wittmaack's operation has not yet been proved, and because in cases of stapedial ankylosis the hearing defect progresses in an uneven manner, with frequent periods of quiescence lasting years, during which there is no measurable increase, the value of any preventive measure will be extremly difficult to assess.)

Bast [2] continues his interesting studies on the embryologic development of the human otic capsule and its possible bearing on the genesis of otosclerosis. The development of the otic capsule as observed by Bast in a study of 61 fetuses and 18 infants is as follows:

The early otic capsule is cartilaginous. Between the sixteenth and twentieth weeks of fetal life the cochlea and the canals reach their maximum size, and ossification of the capsule occurs. At about 16 weeks a center of ossification occurs near the round window, followed quickly by six more centers, the ossification spreading rapidly until by the twenty-first week the capsule is well ossified except for a small area between the vestibule and the cochlea, called the fissular area, and the area medial to the cochlea, where the cartilage continues to grow to form the petrous apex. The ossified cartilage becomes the enchondral layer of the finished otic capsule. A uniform thin inner endosteal layer is laid down, with a thicker outer periosteal layer of bone which continues to increase in thickness.

The fissular area, anterior and medial to the oval window, contains the fissula ante fenestram. Here the enchondral layer of the capsule is very thick, with a thin inner endosteal layer next to the vestibule and a thin outer periosteal layer next to the tympanic cavity.

The fissula ante fenestram is an irregular cleft filled with loose connective tissue, extending from the vestibule to the middle ear. Bast suggests that its function may be to filter the perilymph, which otherwise would stagnate.

Where the fissula is unusually large or shows outpocketings, secondary formation of cartilage commonly occurs, apparently to reduce its size, the cartilage later ossifying. In some cases these cartilage masses become unusually large, and when they ossify later, gnarly unstable bone results, resembling otosclerotic bone. When this gnarly otosclerotic bone extends, it may invo the stapedial joint. The fissular area itself resistant to ossification, but with the increase otosclerotic bone it gradually becomes oblit ated. If the fissula is a structure for the dra age of perilymph its obliteration would result stagnation of the perilymph, and surgical fe estration could reestablish drainage. (Comme Bast's speculation should be given serious co sideration because of his extensive studies on t fissula ante fenestram. There is no reason attribute the improvement in hearing after fe estration to reestablishing drainage of the pe lymph, but the relief of tinnitus and possibly ev the prevention of nerve degeneration might favorably influenced in this way. The drama immediate relief of tinnitus that often follo the operation, with the recurrence of tinni several months later albeit the improvement hearing remains unchanged, suggests that so sort of stagnation of the perilymph might be t cause of the tinnitus.)

Anson and Cauldwell [3] have made a mini and detailed study of the development of t stapes in the human embryo. Most of the stap is derived from the second visceral (hyoid) ar or bar, while part of the footplate and all of t annular ligament are derived from the o capsule. The stapes is unique among hum bones in that it reaches its adult size by t fifth fetal month and its adult form by t seventh. Ossification of the stapes occurs fr a single center of ossification, beginning at weeks. (The ossified stapes is composed large of enchondral bone, with characteristic islands ossified cartilage which persist without chan through the life of the human body. This tis appears to be subject to the pathologic bo change known as otosclerosis, for Guild [4] h found the footplate of the stapes to be the site a primary focus of otosclerosis in a number ears.)

Robb [5] examined the temporal bones of do that had received parathyroid extract and a extract of the anterior lobe of the pituitary co taining the growth-promoting factor and fou histologic changes similar to human otosclero One dog receiving parathyroid extract alo

2. Bast, T. H.: Development of the Otic Capsule: VI. Histologic Changes and Variations in the Growing Bony Capsule of the Vestibule and Cochlea, Ann. Otol., Rhin. & Laryng. **51**:343, 1942; Embryologic Observations Bearing on the Problem of Otosclerosis, Arch. Otolaryng. **36**:816 (Dec.) 1942.

3. Anson, B. J., and Cauldwell, E. W.: T Developmental Anatomy of the Human Stapes, A Otol., Rhin. & Laryng. **51**:891, 1942. Cauldwell, W., and Anson, B. J.: Stapes, Fissula Ante Fenestra and Associated Structures in Man: III. From Embry 6.7 to 50 mm. in Length, Arch. Otolaryng. **36**: (Dec.) 1942.

4. Guild, S. R.: Personal communication to t authors.

5. Robb, J. M.: A Consideration of the Endocri Basis for Otosclerosis, Tr. Am. Laryng., Rhin. & Ot Soc. **47**:64, 1941.

owed no deviations from normal. The author ggests that the pituitary is a prime factor in sclerosis with the parathyroids secondarily in- lved, a constitutional hereditary background termining a locus minoris resistentiae in the yrinthine capsule. (Comment: That there is endocrine basis for otosclerosis is difficult to lieve inasmuch as the disease is confined to the yrinthine capsule, and usually to a small cir- mscribed area in the capsule, in persons who in other respects enjoy normal health. In all imal experiments the "otosclerotic" changes e diffuse, involving not only the temporal bone t other bones, and are therefore hardly com- rable to the focal otosclerosis of the human ing.)

Tamari [6] notes that the pathologic picture of get's disease resembles that of otosclerosis, so t some investigators have regarded oto- lerosis as a retarded or a localized form of get's disease. He describes the temporal bones 3 patients with Paget's disease. In each case th petrous bones were diffusely involved in the ny changes of Paget's disease, i. e., in replace- ent of the normal bone with an irregularly ar- nged spongy bone, the bony changes beginning the areas best supplied with marrow tissue, e enchondral layer of the labyrinthine capsule, e least vascular, being the least susceptible to tack. (Comment: The latter observation ows the essential difference between oto- lerosis and Paget's disease, for in otosclerosis e pathologic bone is first found in the enchon- al layer, and the process chiefly involves this yer.)

Fowler Jr.,[7] having observed 25 patients with perostosis and 9 with discrete exostoses of the ternal auditory canal, of whom all but 4 had en swimmers with an ardent preference for cold ater, irrigated the external auditory canals of uinea pigs with water at 19 C. (67.5 F.). In o case were there sufficient changes in the ternal auditory canal to be of certain experi- ental origin, but in the middle ear on the irri- ated side there were striking changes as com- ared with that on the nonirrigated side. These anges consisted in diffuse formation of new one in the submucosa of the bulla, the amount new bone being more or less proportional to e number of times the cold water had been ed. The changes involved only the periosteal one. (Comment: This unique and interesting servation adds to knowledge on new bone formation in the temporal bone and as such is included in this review. However, since the new bone was entirely periosteal and did not involve the enchondral layer, it is not in any way comparable to otosclerosis, as the author would be the first to point out.)

The literature for 1942 contained 2 articles on the inheritance of otosclerosis. Chumbea [8] presents the pedigree of a man with otosclerosis who married two women with normal hearing, by whom he had 14 children, 3 with otosclerosis. He concludes that otosclerosis is dependent on an autosomal dominant gene but finds that there seems to be some irregularity in the expression of the otosclerotic gene. (Comment: As has repeatedly been pointed out by one of us, and others, histologic otosclerosis without clinical symptoms occurs several times as frequently as does clinical otosclerosis with stapedial ankylosis. Clinical studies to determine the mode of inheritance of otosclerosis are therefore rendered invalid.)

Fowler [9] reports the results of detailed studies of three pairs of identical twins with otosclerosis, including the history, otoscopic and audiometric observations, blood counts, chemical and serologic studies of the blood, basal metabolic rates, fingerprints, photographs, eyegrounds, teeth and cardiograms. He concludes that no significant abnormalities or differences were found which could shed light on the etiologic factors of otosclerosis. (Comment: The fact that in the 8 pairs of identical twins with otosclerosis thus far reported both twins were always affected indicates the predominating influence of heredity in the occurrence of otosclerosis.)

Medical Treatment of Otosclerosis.—Menzel [10] rubbed a freshly prepared ointment of "hormones" and "vitamins" [10a] onto the back of the neck and into the region of the auricle, front and back, once every eight days four to ten times and observed improvements in 39 of 53 cases of middle ear and inner ear deafness. The results are summarized in table 1.

Improvements from hearing a whisper at 10 cm. to hearing one at 8 meters occurred in 1 patient after two treatments. The patient with continuous tinnitus for one year experienced an improvement that was still present

6. Tamari, M.: Histopathologic Changes of the emporal Bone in Paget's Disease, Ann. Otol., Rhin. & aryng. **51**:170, 1942.

7. Fowler, E. P., Jr., and Osmun, P. M.: New Bone rowth Due to Cold Water in the Ears, Arch. Oto- yng. **36**:455 (Oct.) 1942.

8. Chumbea, B. J.: A Pedigree of Otosclerosis, J. Hered. **33**:98, 1942.

9. Fowler, E. P.: Studies of Deafness in Twins: Otosclerosis in Identical Twins; Three Case Histories, Laryngoscope **52**:718, 1942.

10. Menzel, K. M.: A New Form of Treatment for Stubborn Cases of Impaired Hearing, Schweiz. med. Wchnschr. **72**:289, 1942.

10a. The exact sources, the method of preparation and the identity of the "hormones" and 'vitamins" in the ointment are not stated.

after nine months. The author states that the mechanism of action of this treatment is as yet purely hypothetic. (Comment: We cannot accept any treatment of deafness as of value that is not substantiated by audiometric hearing tests, for the reasons repeatedly emphasized in this review. Why rub the ointment on the auricle? The seat of hearing does not lie in the auricle, nor do the venous and the lymphatic drainage from the auricle pass to or through the middle ear or the labyrinth.)

Meyer [11] is shortly presenting his results with suboccipital drainage of spinal fluid in 80 cases of otosclerosis and labyrinthine deafness, but first he wishes to consider the dangers and inconveniences of his treatment. In a study of his 80 cases he concludes that no more inconvenience occurs after this massive drainage of spinal fluid than after the usual diagnostic lumbar puncture.

Woodman [12] regrets the tendency today to diagnose otosclerosis too readily and frequently, for he believes that the usual cause of chronic deafness in the adult is a gradual seepage of bacteria and toxic fluids through an unhealthy eustachian tube to enter the middle ear, leading to degeneration of the auditory nerve. (Both histologic studies and inspection of the stapediovestibular joint during surgical fenestration are proving the correctness of Politzer's statement of more than fifty years ago that the deafness previously believed due to "dry catarrh" of the mucosa of the middle ear is in reality the result of a primary disease of the labyrinthine capsule.)

Gaus [13] believes that otosclerosis is a constitutional disease due to local inferiority of tissue and that the stress and strain of military service could lead to considerable progression of the hearing defect but that any further loss during the ensuing years is due to the natural course of the disease and not to the effects of military service. He notes that any injury to the hearing in otosclerotic patients would be from the effects of noise and trauma of the skull and that some authors regard otosclerotic patients as less liable to the damaging effects of noise than persons with normal hearing. (Comment: With the latter view we agree, for a fixed stapes effectively insulates the organ of Corti and the acoustic nerve not only from speech but from acoustic trauma. It has further been our experience that otosclerotic patients in excessively noisy trades are not bothered by but rather enjoy the noise because of the phenomenon of paracusis Willisii when their hearing has been partially restored by fenestration of the labyrinth, they immediately complain of the terrible, actually painful noise; with their acoustic insulation removed they are no longer protected from the painful and damaging effects of acoustic trauma.)

Surgical Treatment of Otosclerosis.—Lempert, Meltzer and Spector [14] report the results of a series of experiments on regeneration of bone. In the skulls of cats under aseptic conditions defects of varying depth were made with a variety of instruments, and from seven to forty

TABLE 1.—*Results Reported by Menzel [10] as Obtained with an Ointment of Vitamins and Hormones*

	Otosclerosis	Labyrinthine Deafness	Residue of Middle Ear Disease	Tinnitus
Markedly improved	7 (50%)	10 (41.6%)	4 (28.5%)	1 (100%)
Moderately improved	6 (43%)	4 (16.6%)	7 (50%)	
Unchanged	1 (7%)	10 (41.6%)	3 (21%)	

six days after operation the bones were examined histologically.

When the defect was made with the dental drill and a no. 12 gold-polishing burr, at seven days it was filled with connective tissue, and some of the fibroblasts showed eccentric nuclei indicating beginning transformation into osteoblasts. At fourteen days a defect made with the same burr showed active and well advanced new bone formation, with trabeculae of new bone growing from the margins of the old and showing a clear and sharp line of demarcation between the old and the new bone.

Defects examined twenty-one days after they were made with the finishing burr, with a cutter with a sharp no. 10 dental burr and a 5/8 inch (1.6 cm.) disklike circular saw, respectively showed active and advanced new bone formation which nearly obliterated some of the defects made with the sharp instruments and was less advanced in the defects made with the finishing burr. After thirty-nine and forty-six days the defects made with a polishing or dull burr showed

11. Meyer, M.: Factors to Be Considered in Major Suboccipital Puncture as Recommended by the Author in Certain Forms of Deafness: Problems of Cerebrospinal Fluid Pressure and the Ear, Pract. oto-rhino-laryng. **4**:93, 1942.

12. Woodman, E. M.: The Deaf Adult: Problems of Diagnosis and Treatment, Practitioner **148**:342, 1942.

13. Gaus, W.: Evaluation of Effects of Military Service on Otosclerosis as a Severe Disability, Deut. Militärarzt **6**:104, 1941.

14. Lempert, J.; Meltzer, P. E., and Spector, B. Bone Repair Following Defects Made in the Skull of Cats by Means of Different Instruments, Arch. Otolaryng. **36**:473 (Oct.) 1942.

new bone. In all cases the defect on the left side was left unburnished while the one on the right was burnished with a smooth steel ball-shaped burr. In no case was there a significant difference between the burnished and the unburnished lesion.

The authors conclude that, given sufficient time, growth of new bone will take place in a lesion made with any type of instrument. The finishing and burnishing burrs tend to retard and inhibit osteogenesis, but in no case will they prevent it. They thus disagree with the conclusions reached by both Canfield and the Guggenheims. (Comment: Canfield found active regeneration of bone after two weeks when the defect was made in the skull of a cat with a sharp instrument and no regeneration of bone when the defect was made with a dull instrument, and concluded that the type of instrument used to make the defect is more important than the tissue used to cover it. The Guggenheims found lively and widespread regeneration of bone eighteen days after making defects in the skulls of rabbits whether they used a sharp or a dull instrument, and after two months and two days the fistula made with the sharp instrument showed less regeneration than the one made with the dull burr. They concluded that the type of instrument makes no difference in the resulting regeneration of bone. Lempert and his co-workers, in a more careful and extensive study, found that the type of instrument makes some difference but certainly is not the whole answer as to why the labyrinthine fistulas as now made remain open as often and as long as they do. This valuable contribution to knowledge of the basic processes of healing of bone is beautifully illustrated with excellent photomicrographs that clearly demonstrate the histologic changes. The answer as to why surgical fistulas remain open as well as they do must lie in part in the fact that the enchondral layer of the labyrinthine capsule differs from the periosteal bone, which was used by these authors and by Canfield and the Guggenheims, in its sluggish response to trauma as compared with the active response of periosteal bone.)

Campbell [15] presents a follow-up report on the 30 fenestrations reported the year before, these first 30 cases showing no essential change in the interim. Proper operative indications were present in 21 patients. Thirteen, or 62 per cent, of these have a hearing improvement of more than 14 decibels for the three speech frequencies, the average improvement for this group being 23.6 decibels. The 8 patients in whom the operation proved unsuccessful showed an average loss of 3 decibels.

Of 23 additional patients operated on who had been observed for periods of eight months or longer, proper indications were present in 22; 13, or 59.9 per cent of these, showed an improvement of more than 12 decibels; in 9 the operation was unsuccessful.

All of the first 42 patients were operated on according to the technic described by Lempert; the last 11 were operated on by a combined postauricular and endaural incision, the bone work being performed through the postauricular incision. Of the last 11 subjects, 4, or 36.4 per cent, showed improvement, compared with 8, or 66.7 per cent, of the preceding 12 patients operated on by Lempert's endaural technic.

Summing up the results in the 53 cases reported by the author, one notes that 43 were suitable, and in 60.5 per cent of these the hearing for the speech frequencies was improved more than 12 decibels. In 5 cases the tympanomeatal membrane was badly lacerated, remnants being placed over the fistula in 3, and a skin flap dissected from the wall of the anterior canal being used to cover the fistula in 2. In these 5 cases the hearing improvements for the speech frequencies were especially great, amounting to 37, 25, 35, 36 and 34 decibels as end results.

Postoperative complications were present as follows: in 4 cases, facial paralysis, with complete recovery in four to six weeks; in 11 cases, infection of the cavity operated on, controlled with sulfathiazole powder; in 4 cases some constriction of the external auditory canal; in 2 cases, serous labyrinthitis; in some cases delayed and extremely slow healing.

A summary of the results in the cases in which improvement was noted for more than eight months after operation is given in table 2.

(Comment: The most conservative critics of the fenestration operation will have trouble finding flaws in Campbell's commendably complete report, which demonstrates audiometrically substantiated significant and lasting hearing improvements in half of his cases. Some of Campbell's cases are particularly noteworthy: case 1 with a 37 decibel gain three years and nine months after operation; case 27 with a 38 decibel gain one year and nine months after operation, bringing the hearing to 6.6 decibels of normal; case 53 with a 33.4 decibel gain eight months after operation, bringing the hearing to 11.6 decibels of normal. These truly phenomenal and almost miraculous results are a challenge to all otologists to equal or surpass them by any form of medical or surgical treatment for otosclerosis. The fact that in a third of his properly selected

15. Campbell, E. H.: Fenestration of the Labyrinth: A Report and Analysis of Operated Cases, Laryngoscope **52**:593, 1942.

cases Campbell failed to obtain a significant lasting improvement, while in others he witnessed a definite but disappointingly small gain as contrasted with the dozen or so truly brilliant results, indicates that the Lempert fenestration technic gives results that still leave much to be desired. Nevertheless there can be no question but that the fenestration operation as evolved by Holmgren, Sourdille and Lempert represents one of the great, truly momentous contributions to otology.)

Shambaugh [16] presents in tabulated form the complete data on 170 consecutive fenestrations, describes a modification of the Lempert technic and compares his results from four different modifications of the technic.

TABLE 2.—*Results of Surgical Fenestration in Cases in Which Improvement Was Still Present After Eight or More Months, as Reported by Shambaugh* [16]

Case	Months Since Operation	Average Loss of Hearing After Operation, Decibels	Average Improvement in Hearing, Decibels
1	45	31.6	+37
6	24	60	+16
8	14	51.6	+25
11	36	20	+22.3
12	36	73.3	+23.3
17	31	43	+14
18	21	45	+18.3
21	24	48.3	13.3
22	23	41.6	+20
25	20	18.3	+24.7
27	21	6.6	+38
29	12	22.2	+35.5
30	4	23.3	+18.7
32	16	36.6	+19.4
33	17	26.6	+24
34	17	40	+16 3
35	17	35	+29 6
36	9	38.3	+25
37	12	65	+12.6
38	14	46 6	+14
39	12	35	+13
43	12	26	+36
46	10	28.3	+13.3
47	10	46.6	+12.7
52	9	23 3	+24
53	8	11.6	+33.4

In a study of the 26 cases in which the improvement was lost through closure of the fistula, Shambaugh found that closure rarely begins later than the fifth postoperative month and is complete in most cases within six months. He concludes that at least six months must elapse before the result of the operation can be judged.

The author believes that the relative merits of different surgical technics to improve the hearing of the patient with otosclerosis can be assessed in only one way — by a careful analysis and comparison of the hearing results. He lists the minimum data that should be reported to permit evaluation of the results of any therapy for chronic deafness:

1. Every case in which the operation was done should be reported.
2. Hearing should be measured audiometrically before and after operation, the hearing change for the three speech frequencies being averaged in terms of decibels gained or lost. A change of less than 10 decibels must be regarded as within the limits of variability and as of no consequence.
3. At least six months should elapse before the results of the surgical intervention are judged.
4. There should be adequate controls. The best control is the ear which has not been operated on, and its hearing change should be recorded.
5. All complications and poor results should be recorded.

Following these criteria, the author compares his results from four different fenestration technics (table 3):

1. The original Lempert technic.
2. The original Lempert technic modified by the addition of irrigation during the making of the fistula. The irrigating apparatus provides a constant stream of warm sterile isotonic solution of sodium chloride across the bony horizontal semicircular canal, to wash away all particles of bone dust and to keep the field immaculately clean and free from blood.
3. The second technic with the further addition of the use of a binocular dissecting microscope to provide greater magnification while the fistula is being made.
4. Lempert's nov-ovalis technic with the addition of irrigation and the use of a microscope.

(Comment: The minimum data listed by the author as essential to permit evaluation of the results of therapy of chronic deafness are not original with him but are essentially those suggested by Fowler Jr. in the latter's excellent "Critique of Surgical Treatment of Deafness" and those followed by Campbell and Hughson in their excellent and complete reports of results of surgical therapy for chronic deafness. It is to be hoped that all future reports of therapy of deafness will adhere closely to these criteria following the examples set by Fowler Jr. and by Campbell and Hughson.)

Hall [17] describes a technic for the operative treatment of otosclerosis in which the head of the malleus is removed, a fistula is made in the horizontal semicircular canal and a skin flap, taken from the roof of the external auditory meatus and attached to the drum membrane, is used to cover the fistula. An operating microscope is used, and in recent cases amnioplastin has been placed beneath the flap to prevent fibrous tissue forming between the membranous labyrinth and the flap. It is as yet too early to judge the results in these cases.

16. Shambaugh, G. E., Jr.: Surgical Treatment of Deafness, Illinois M. J. **81**:104, 1942; Otosclerosis: Results with the Fenestration Operation After Three and One-Half Years, J. A. M. A. **119**:243 (May 16) 1942; A Modified Fenestration Technic, Arch. Otolaryng. **36**:23 (July) 1942; Surgical Treatment of Otosclerosis: Comparative Analysis of Results Using Different Technics, Ann. Otol., Rhin. & Laryng. **51**: 817, 1942.

17. Hall, I. S.: Progressive Middle Ear Deafness, Edinburgh M. J. **49**:352, 1942.

Of 17 patients operated on with "improved ethods" (Hall does not specify the improvements), 2 were operated on less than six months ior to reporting, so that it is too early to judge e results, 10 show hearing improved and 5 aring unchanged. The number of decibels of aring gained is not specified, but the audiogram of a typical case shows an average gain r the speech frequencies of 22 decibels, bringg the hearing for speech to the 40 decibel level. total of 31 patients have been operated on, but e results are not clear from the table presented the author. (Comment: Apparently he emoys Lempert's one stage fenestration technic, ough he does not give credit to Lempert or to ourdille for this technic. The article is of parcular interest since except for the report of 2 ses in which the operation was unsuccessfully mployed by Gasse, in 1939, it is the first report results published by any one outside this

estration if the author's figures tell the whole story. However, the tests with the hearing aid were apparently made with pure tones. It is possible that were there some satisfactory way of measuring the hearing for speech with the hearing aid as compared with the hearing for speech after fenestration, the conclusions would be different. It is my experience that the many patients who have been treated by fenestration and who previously used a hearing aid for several years, even as long as twenty years, almost without exception state that they now hear better without their aid — provided the fenestration operation resulted in an average gain of 20 decibels or more for the speech frequencies. However, the surgeon should not be satisfied with the patients' statements; it is up to him to accept Berry's challenge and prove by objective tests if he can that the patients hear better after fenestration.)

TABLE 3.—*Comparison of Results from Four Different Fenestration Technics, According to Shambaugh*[16]

	Total Operations	Total Patients with Hearing Improved	Percentage with Gain of			Percentage with Hearing Unchanged	Percentage with Hearing Worse
			10 to 19 Decibels	20 to 29 Decibels	30 to 47 Decibels		
Original Lempert technic	15	4 (27%)	27	0	0	53	20
Irrigation and Lempert technic	29	22 (76%)	21	34	21	24	0
Irrigation and use of microscope with Lempert technic	55	43 (78%)	30.9	29.1	18.3	20	1.8
Irrigation and use of microscope with Lemper nov-ovalis technic	32	28 (88%)	19	47	22	9	3

untry using Lempert's procedure. Unfortutely, the author does not present the minimum ta needed to evaluate his results.)

Berry[18] raises the question of whether fenstration can do as much for the patient with otoclerosis as a well fitted hearing aid. He takes e average gains in series of selected cases in hich fenestration was done—25.7 decibels reorted by Shambaugh, 24.3 decibels reported by empert and 21.8 decibels reported by Campbell —and contrasts these with an average gain of 43 ecibels reported in a series of selected cases with earing aid fittings. The latter figure was obined from 13 patients fitted with the Maico earing aid using a vacuum tube air conduction nstrument. The Council on Physical Therapy of e American Medical Association in a study of ght of the commonly used aids reported an verage amplification of 30 decibels. From these ata the author concludes that a well fitted modrn hearing aid can give greater amplification an fenestration, to a wider group of hard of earing persons, at less expense, pain and worry spite of the nuisance factor. (Comment: This s an excellent and very valid criticism of fen-

Hill,[19] discussing the treatment of chronic progressive deafness, states that for the majority of patients with established deafness, correction by amplification will be preferable to operative treatment. Operative treatment may be suggested to those who, after critical study, meet all the requirements laid down as essential to successful fenestration, provided they fully understand its present status. (Comment: The candidate for fenestration should understand fully the possibilities of failure as well as of success, the discomforts, the inconveniences, the dangers and particularly the occasional disturbance of equilibrium that may follow the operation. Taking all of this into account, some will prefer and should get an electrical hearing aid; others will be willing to go through the risks and possibilities of failure for the reasonable promise of success.)

CHRONIC ADHESIVE DEAFNESS

Loch[20] carried out experiments on himself to determine the effect on hearing of acute occlusion

18. Berry, G.: The Case for the Hearing Aid, aryngoscope **52**:615, 1942.

19. Hill, T. T.: The Changing Conception of the Management of Chronic Progressive Deafness, Ann. Otol., Rhin. & Laryng. **51**:653, 1942.

20. Loch, W. E.: The Effect on Hearing of Experimental Occlusion of the Eustachian Tube in Man, Ann. Otol., Rhin. & Laryng. **51**:396, 1942.

of the eustachian tube. A small rubber bag was inserted into the nasopharynx through one nostril and inflated until breathing through both nostrils was completely obstructed. Audiograms were made at frequent intervals over one and a quarter hours, with the following results in the right ear: At ten minutes after occlusion was begun, there was a slight improvement of 5 decibels for all frequencies except 4096, the hearing for which had improved 20 decibels. At twenty minutes there was a 5 decibel drop for some of the high frequencies. At forty-five minutes the audiogram showed a drop of 5 to 15 decibels for all of the frequencies above 512 compared with the ten minute audiogram. At sixty minutes there was a drop of 5 to 20 decibels for all frequencies above 256, greatest for the highest tones of 8192 and 10321 vibrations. At seventy-five minutes there was a 5 decibel drop for the tones 64, 256, 512 and 1024; for the higher tones there was a 10 to 20 decibel drop.

Immediately after the balloon was removed and the ear inflated by the Valsalva procedure, the hearing returned to normal for all but 4096 and 5793, for which it showed a 5 decibel loss, but after a second ventilation the hearing for these two tones returned to normal.

Similar tests on the left ear gave similar results.

Loch concludes that an uncomplicated tubal occlusion causes a hearing loss for high tones chiefly, sometimes followed by a loss for low tones. The prompt recovery on inflation proves that this loss was due solely to changes of pressure in the middle ear and not to secondary edema in the organ of Corti or the round window membrane.

Loch [21] then produced sudden marked increase and decrease in intraxympanic pressure by Valsalva's procedure, roughly estimating the change in pressure by a manometer in the external auditory canal and another in the nostril. Audiograms showed a marked hearing loss for low tones, as much as 20 to 30 decibels, with considerably less impairment for the higher tones 2048 and 4896. For the highest frequencies 10321 and 13004, however, the greatest loss of all was shown, 30 to 35 decibels, as the result of negative pressure, with no change or even a slight gain on positive pressure. (Comment: The author does not venture an explanation for the markedly different type of hearing curve obtained when the eustachian tube was blocked for an hour as compared with that obtained after a sudden marked decrease in intratympanic pressure. These experiments are of fundamental interest and importance in adding to the present incomplete knowledge of the mechanism of hearing impairment due to tubal occlusion.)

Decker [22] believes that blockage of the eustachian tube in childhood plays an important role in the development of chronic progressive deafness in adult life. After repeated attacks of tubal occlusion the membranes of the tube become thickened and the lumen narrows, resulting in progressive loss of hearing. Decker advocates for the acute stage of tubal occlusion application of shrinking solutions to the orifice of the tube and of mild antiseptics and astringents to the nasopharynx. Later the cause should be removed by roentgen irradiation of the lymphoid tissue of the nasopharynx. Other therapeutic measures include desensitization in cases of allergy, breaking up of adhesions and scars about the tubal orifice by digital manipulation and massage, bougie treatment of strictures of the tube, prevention of colds and attention to the general health. (Comment: It is our belief that while tubal occlusion is the most common cause for slight to moderate hearing losses in childhood and for many cases of slight to moderate hearing losses in adult life the pathologic changes in the middle ear are essentially reversible, for we have seen the hearing return to within normal range from a 30 to 40 decibel loss of twenty years' duration as the result of removal of hypertrophied adenoids. Perhaps in occasional cases a long-standing untreated tubal occlusion will result in some adhesive changes in the middle ear, but the hearing impairment is not progressive in the sense that there is progressive deterioration to a profound loss such as one sees in otosclerosis. Progressive conduction deafness in adult life is due to otosclerosis in the great majority of cases, and treatment of an imagined tubal occlusion will have no effect on the loss of hearing. As a matter of experience, true stenosis of the eustachian tube is exceedingly rare; temporary occlusion of the tube from adenoid hypertrophies or from allergic or inflammatory edema is common; sometimes it is caused by carcinoma of the nasopharynx. The clinical diagnosis of impairment of hearing due to tubal occlusion is made easily and accurately by simple inflation of the tube. An immediate demonstrable improvement in hearing indicates that there has been negative pressure in the middle ear due to tubal occlusion. The auscultation of fluid in the middle ear on catheteral inflation is important corroborative evidence of tubal occlusion of severe degree. The treatment of tubal occlusion in our practice con-

21. Loch, W. E.: Effect of Experimentally Altered Air Pressure in the Middle Ear on Hearing Acutely in Man, Ann. Otol., Rhin. & Laryng. **51**:995, 1942.

22. Decker, R. M.: Relation of the Eustachian Tube to Chronic Progressive Deafness, Arch. Otolaryng. **36**:926 (Dec.) 1942.

ts in repeated catheteral inflations until mois-
re in the middle ear as demonstrated with the
scultation tube has cleared up and there is no
ger an objective improvement in hearing after
lation. When there is an allergic factor
sually with nasal allergy), or adenoid hyper-
phy, or a thick mucoid secretion in the middle
that fails to absorb after several inflations,
atment of the allergy, surgical or roentgen
removal of the adenoid tissue, or paracentesis
d inflation of the middle ear are done. The
gnosis of hearing impairment due to tubal
clusion is excellent. The prognosis of hear-
impairment due to otosclerosis or to adhe-
ns which are the residue of infantile otitis,
ere suppurative otitis or primary degeneration
the acoustic nerve is poor, and inflation
ssage, bouginage or irradiation for an imagi-
ry tubal occlusion will not avail; treatment of
s type will only lead to discrediting of these
erapeutic measures.)

Crowe [23] and his co-workers report the results
radon treatment of the nasopharynx in 152
ildren with impaired hearing and lymphoid
ergrowth of the tubal orifices. After irradia-
n 89.5 per cent of these children showed nor-
l tubal orifices, and only 10.5 per cent still
d enough lymphoid tissue to require further
atment. In a control group of children with
rmal hearing and hypertrophied lymphoid tis-
e spontaneous regression of the lymphoid tis-
e occurred in 31 per cent and partial regres-
n in 15 per cent; in almost 10 per cent there
s an increase in the lymphoid tissue. The
erage number of treatments was four; a few
tients have had more than six, and some of
se needing further treatment had a regrowth
lymphoid tissue after it had once undergone
gression.

The effect on the hearing was more difficult to
aluate: About one third of the ones having
aring defects limited to the high tones showed
finite improvement in acuity of hearing for
ese tones.

The authors conclude that the evidence is in
vor of radon treatment of hyperplastic lymphoid
sue in the nasopharynx in childhood, and that
e impairment for high tones in the ears that
proved was caused by lesions of the middle ear
at regressed after treatment of the nasopharynx
d favorably influenced ventilation of the mid-
e ear. (Comment: Presumably the high tone
ss in the patients whose ears did not improve
s not due to impairment of the ventilation of the middle ear, indicating that further diagnostic measures should be employed to determine which patients can and which cannot be helped by irradiation of the nasopharynx. Perhaps diagnostic inflation as described in the foregoing comment on the article by Decker [22] would help the otologist to diagnose more accurately high tone loss due to tubal obstruction.)

Emerson, Dowdy and Heatly [24] describe a new applicator for applying radium to the nasopharynx—a semiflexible steel wire, 13.4 cm. long, having one end threaded to hold a cap containing two 12.5 mg. radium needles. Two such applicators are applied intranasally, one to each eustachian tube, for twelve minutes, delivering 5 milligram hours, or about one third of an erythema dose. In the majority of patients the eustachian tube will appear normal and open by the end of four weeks, and the hearing will approach normal. Some will need further treatment, but three months is allowed to elapse between treatments. The authors state that deafness as detected by the audiometer and the presence of eustachian adenopathy are the two main criteria for treatment. Six cases picked at random out of almost 100 in which the treatment was given are presented, with audiograms made before and after treatment. (Comment: Of the 6 cases, 5 had shown a considerable loss for all tones, and only 1 had shown only the high tone loss stressed by Crowe and his co-workers. The hearing improvements in these cases are impressive, with a restoration of approximately normal hearing in each case, from an average loss of 20 to 40 decibels for the speech frequencies. Even when the impairment had been present for several years the hearing was restored to normal by removing the tubal obstruction. This coincides with our experience: Tubal obstruction itself does not lead to permanent progressive adhesive changes in the middle ear, even when the obstruction is of many months' or years' duration.)

Farrior and Richardson [25] describe an applicator for applying radium to the nasopharynx consisting of a pliable copper wire attached to a 50 mg. radium capsule. The brass capsule has a wall thickness of 0.8 mm., equivalent to 0.2 mm. of platinum. It contains five standard 10 mg. radium needles, each with a wall thickness of 0.3 mm. of platinum, so that there is a total of 0.5 mm. of platinum equivalent, enough to remove the caustic beta rays. One applicator is used in each side of the nasopharynx for one hour (50

23. Crowe, S. J.; Guild, S. R.; Langer, E.; Loch, E., and Robbins, M. H.: Impaired Hearing in hool Children (Radon Therapy of Nasopharyngeal mphoid Tissue). Laryngoscope **52**:790, 1942.

24. Emerson, E. B., Jr.; Dowdy, A. H., and Heatly, C. A.: Use of Radium in Treatment of Deafness by Irradiation, Arch. Otolaryng. **35**:845 (June) 1942.

25. Farrior, J. B., and Richardson, G. A.: A Nasopharyngeal Radium Applicator, Arch. Otolaryng. **35**: 811 (May) 1942.

milligram hours). No undesirable effects have occurred. (Comment: The dose used by these authors is ten times as large as that used by the authors of the preceding article. The smallest dose that would accomplish the regression of hypertrophied lymphoid tissue would appear to be the best.)

Langer [26] studied the 1,365 school children examined by Crowe and his co-workers, noting the relation between hyperplastic nasopharyngeal lymphoid tissue and the hearing, the condition of the teeth, the height, the weight, the shape of the face and head and allergic conditions. He concluded that there was no relationship between hypertrophied lymphoid tissue in the nasopharynx and these factors except that there was a tendency for large adenoids to occur in children with bad teeth, but he could not conclude that there was any definite correlation between the two conditions. (Comment: Clinically it has been our impression that lymphoid hypertrophy is more frequent in allergic children, but Langer did not find this to be so.)

Hunt [27] presents a statistical analysis of 1,080 hard of hearing school children examined otologically. There were 401 cases of suppurative healed otitis (adhesive otitis without discharge at the time of examination), 394 cases of nonsuppurative healed otitis (catarrhal otitis media), 125 cases of active suppurative chronic otitis and 54 cases of nerve deafness. As etiologic factors responsible for the aural condition there were: hypertrophy of lymph tissue in 100 cases; pathologic alteration of the turbinates in 189 cases; obstruction by a deviated septum in 15 cases. (Comment: Programs for the prevention of deafness have centered largely on school children, and rightly, because the types of deafness that are amenable to prevention or treatment are those occurring especially in school children, namely, obstruction of the eustachian tube by hypertrophied lymphoid tissue and suppurative otitis media. The first step in any effective preventive program is to detect the hard of hearing child, and this is now being well done in certain cities and states by routine testing of the hearing of school children. The second step is to see that appropriate otolaryngologic treatment is carried out when indicated, and here at present is the chief obstacle to an effective program for the prevention of deafness, for there are too few otologists properly prepared to do this work, and there is no effective machinery for directing t children to these properly prepared otologis Otologists should take the lead in organizi whatever system can best direct the school ch dren needing treatment to those who can a will carry out the required measures, with effective follow-up system to encourage pare to carry out the recommendations.)

Fowler Jr.[28] states that one kind of deafn that yields to treatment in the adult as well as the child is that caused by obstruction of t eustachian tube. Inflation causes temporary r lief, but the infection in the nose, the sinus and the nasopharynx must be treated. children removal of hypertrophied adenoids of paramount importance. Radiotherapy hypertrophied lymphoid tissue in and about t eustachian tube achieves cures most often children, but a few adults have been materia benefited. The author used a special applicat for the insertion of radium element into the nas pharynx. The larger series of patients w treated with roentgen rays. The author sugge that the main effect was the reduction of infla mation in the subepithelial tissue of the nose w reduction of secretion, and not direct destructi of lymphoid tissue by roentgen rays. (Co ment: Whether treatment of the nasopharyn with radium emanations [radon], with radi element or with high voltage roentgen rays the most effective for hypertrophic lympho tissue causing subacute or chronic tubal occlusi has not yet been determined. We have u roentgen rays, chiefly because of convenien and have been satisfied with the results. O experience coincides with that of Fowler J that hypertrophied lymphoid tissues are an i portant cause of tubal occlusion, especially the child, but that hypersecretion and bog nasal and tubal mucosa are also an import cause in children and are a more frequent ca of chronic tubal occlusion in adults than hyp trophied lymphoid tissues. In many patien we believe, the occlusion is on an allergic ba and will respond to allergic management.)

Meltzer [29] urges the use of the modified radi mastoid operation for chronic suppurative oti limited to the epitympanic space, with the pa tensa intact, in order to preserve the hearing. is a mistake to approach cases of this type w the sole idea of getting rid of the dischar paying little attention to the preservation of hea ing. (Comment: In a considerable proporti of cases of chronic suppurative otitis media t

26. Langer, E.: Impaired Hearing in Children and Size of Adenoids in Relation to Some Anthropometric Data and to Condition of Teeth, Ann. Otol., Rhin. & Laryng. **51**:931, 1942.

27. Hunt, W. M.: What Is Being Done for the Deafness of School Children? Laryngoscope **52**:230, 1942.

28. Fowler, E. P., Jr.: Nonsurgical Treatment Deafness, Laryngoscope **52**:204, 1942.

29. Meltzer, P. E.: Hearing in Chronic O Media, Ann. Otol., Rhin. & Laryng. **51**:727, 1942.

s tensa is intact and the disease is confined to attic with cholesteatoma formation. Irrigations of the attic suffice to remove the infected bris and render the cavity clean, dry, odorless inactive in the majority of cases. When the c cholesteatoma is larger and more inaccessible, irrigations alone will not produce a dry ear, the radical mastoid operation as modified by ndy should be done.)

Pohlman [30] regards the organ of Corti in the ernal ear as being acoustically insulated inst air vibrations by virtue of its immersion a liquid medium enclosed in a bony capsule h two resistant windows. The sound-conting mechanism overcomes this acoustic ination by transferring air vibrations to the uid of the inner ear. Conduction deafness may defined as the normal acoustic insulation of internal ear. It amounts to a maximum of ± 5 decibels. This acoustic insulation may overcome in four ways:

1. By restoring function to the intact middle ear apparatus, as by inflation of an occluded eustachian tube.
2. By increasing the intensity of the sound, as with an electrical hearing aid.
3. By reducing the acoustic insulation by making a large window covered by a thin membrane (the fenestration operation).
4. By replacing the damaged or lost middle ear apparatus with a prosthesis.

The prosthesis devised by Pohlman resembles sound-conducting apparatus of the bird, ich consists of a diaphragm coupled to the oval ndow by a straight rod called the columella. hlman makes the diaphragm of his prosthesis m a no. 00 gelatin capsule with the ends nched out and a bit of moistened Cargile mbrane between the two halves. A nylon ment 0.022 inch (0.056 cm.) in diameter is through a needle hole in the diaphragm, the of the filament being fused to form a bead. The contact area over the oval or the round ndow is located by probing with a long slender m of bamboo tipped with a bead of sealing x, until the patient suddenly hears louder. e prosthesis is then introduced.

Kobrak [31] in a series of animal experiments ds that the full effect of the intratympanic scle contractions does not come into force til the damping effect has passed its climax is nearly over. He suggests that in addition the protective mechanism of damping against sounds of dangerous intensity, there is a "corrective damping" of sounds of lesser intensity and that hyperactivity of the reflex arc may contribute to impairment of hearing in certain cases. (Comment: This is a novel and interesting theory without any clinical substantiation as yet.)

AVIATION AND SUBMARINE DEAFNESS

McGibbon [32] explains by means of simple models the response of the eustachian tube to changes in atmospheric pressure. With ascent and decreasing pressure the air in the middle ear escapes easily and automatically through the normal eustachian tube approximately every 435 feet (132.5 meters). In descent, however, the tube does not open passively, muscle action being required. If the external pressure becomes 90 mm. of mercury greater than the intratympanic pressure, the eustachian tube "blocks" and cannot be opened even with muscular action. It is then necessary to open the tube by forced ventilation. The author emphasizes the importance of early treatment of the "blocked tube," before changes have occurred in the tubal mucosa that may prevent forced inflation.

McGibbon [33] uses the term "aviation pressure deafness" instead of the term "aero-otitis," used in America. In 100 cases of aviation pressure deafness the deafness was the most constant and almost invariable symptom; pain was present in half the cases; tinnitus and vertigo were symptoms in a small proportion. Otoscopically, 47 patients had invaginated drum heads, 6 attic congestion, 8 congestion of the entire tympanic membrane, 6 effusion into the tympanum and 7 rupture of the tympanic membrane.

Ventilation of the middle ear within a short time after the onset of symptoms may be effected by Valsalva, Politzer or catheteral inflation, with relief of symptoms. If inflation is neglected for one or two hours, the tube becomes impermeable to air (even at higher pressures), fluids or sounds. The nature of the changes in the tubal mucosa responsible for this impermeability can only be surmised.

Fenton [34] reviews the different effects of flight on the ear as follows:

Exposure to noises causes a sudden drop in hearing around 4,096 vibrations.

0. Pohlman, A. G.: Acoustic Insulation of the chlea and Its Relation to Fenestration Operation and ddle Ear Prosthesis, Tr. Pacific Coast Oto-Ophth. **27**:27, 1942.

1. Kobrak, F. W.: A Study of the Activities of the ratympanic Muscles, J. Laryng. & Otol. **57**:418, 2.

32. McGibbon, J. E. G.: The Nature of the Valvular Action (Passive Opening) of the Eustachian Tube in Relation to Changes of Atmospheric Pressure and to Aviation Pressure Deafness, J. Laryng. & Otol. **57**:344, 1942.

33. McGibbon, J. E. G.: Aviation Pressure Deafness, J. Laryng. & Otol. **57**:14, 1942.

34. Fenton, R. A.: Otology and Aviation, Ann. Otol., Rhin. & Laryng. **51**:333, 1942.

Acute aero-otitis media, due to descent with failure of the eustachian tubes to open, causes low tone deafness of varying degrees. Persons with acute rhinopharyngeal infections should not be permitted to fly.

Airsickness is due to a combination of excessive labyrinthine stimulation, visual impressions and visceral disturbances.

Alcoholic indulgence leads to recklessness; it also produces anoxia of tissues, which appreciably lowers the aviator's ceiling.

The indiscriminate local use of ephedrine and benzedrine in the nose is undesirable because of the subsequent secondary engagement of the nasal and tubal mucosa.

"Blacking out" at high altitudes is due to anoxemia and not to nitrogen gas embolism.

Persons with high blood pressure or impaired coronary circulation should not fly.

Campbell [35] lists seven otolaryngologic problems of military aviation:

1. Selection. Any average intelligent healthy normal person possessing an average nose of normal function, average ears and hearing, eustachian tubes and sinuses without obstruction, and a balance mechanism within the normal range can become a good airman.
2. Hearing. Since the information gained through the ears is absolutely necessary for operational flight, it becomes a great problem to help the air crew to keep their hearing at a high degree of efficiency at all times. Fortunately, most hearing changes from flight are transitory, and after complete ventilation of the middle ear and a few hours of quiet the hearing usually returns to normal.
3. Aero-otitis media may take three to twenty-one days to disappear completely, infection being rare.
4. Airsickness. This remains in the realm of selection, though there is a certain amount of conditioning possible.
5. Aerosinusitis. This is similar to aero-otitis. Those with chronic sinusitis, acute rhinitis or sinusitis or obstructed sinuses should not fly.
6. Speech at high altitudes is difficult because of the lowered density of the air passing through the vocal cords.
7. Anoxia at high altitudes affects the higher centers and interferes with the interpretation of sounds.

Carson [36] discusses aero-otitis and auditory fatigue from exposure to excessive noise, noting that both are purely transient. He also notes that in learning to fly one must learn to ignore most of the labyrinthine, tactile, visceral and deep muscle stimuli and substitute therefor a c pletly visual adaptation. Labyrinthine sensati especially serve to confuse the flyer.

Simpson [37] in discussing aero-otitis states it may be complicated by a severe and perma form of nerve deafness. Testing of the laby thine apparatus by rotation has been found little help in the selection of personnel and been given up.

Shilling and Everley [38] observed 152 case aero-otitis media that occurred in subma escape training. Prior to entering the water e man was subjected to 50 pounds (22.7 Kg.) increase of air pressure in the recompress chamber. In most cases equalization of p sure in the middle and the outer ear took pl spontaneously through the eustachian tube, some found it necessary to swallow or yawn, still others to carry out the Valsalva proced to achieve the rapid equalization necessary. H ever, in some cases none of these measures effective, and because of severe pain in the the subject had to be returned to normal p sure.

Treatment consisted of instilling a solu containing ethyl aminobenzoate and antipyrin glycerin in the external ear and spraying an ep drine inhalant into the nose. Catheteral inflat of the eustachian tube gave no more relief t the "auralgan" ear drops.

Prevention depends on the elimination of with acute or chronic infections of the up respiratory tract or marked nasal obstruction polyps or a deviated septum. The prelimin inhalation of helium-oxygen mixtures is of doubted value, and education in the technic self inflation of the tubes is of distinct va (Comment: The value of "auralgan" ear dr must be purely symptomatic, to decrease the p and could not help to relieve the tubal occlusi

Almour [39] describes the "blocked ear" of caisson worker, occurring during compres or decompression, due to tubal occlusion, usu from the common cold. In the majority of ca without perforations hearing returns to norm the average time being sixteen days, the short four days, the longest twenty-eight days. (C ment: The "blocked ear" described by the aut is apparently identical with aero-otitis media.

35. Campbell, P. A.: Otolaryngologic Problems of Aviation in World War II, Ann. Otol., Rhin. & Laryng. **51**:293, 1942.

36. Carson, L. D.: Otolaryngologic Aspects of Aviation, Laryngoscope **52**:704, 1942; Otolaryngologic Problems Occurring in Fliers, J. A. M. A. **120**:4 (Sept. 5) 1942.

37. Simpson, J. F.: A General Survey of rhinological Considerations on Science Aviation. Laryng. & Otol. **57**:1, 1942.

38. Shilling, C. W., and Everley, I. A.: Aud Acuity in Submarine Personnel: III. Aero-Otitis M in Submarine Escape Training, U. S. Nav. M. **40**:664, 1942.

39. Almour, R.: The "Blocked Ear" of the Cai Worker, Laryngoscope **52**:75, 1942.

fficult to understand how it could occur on mpression, in which the increased pressure he middle ear should be equalized without culty by the escape of air into the naso- ynx.)

PRIMARY NERVE DEAFNESS

erve Deafness from Acoustic Trauma.—ff [40] observed three types of pathologic nge in the cochleas of 3 patients who had exposed to acoustic trauma. The first ent, a woman of 29, had shot herself in the l. The tectorial membrane was a thin line utinated to the indistinguishable cells of the an of Corti. Reissner's membrane was in- The second patient, a 52 year old mother, a fracture of the footplate of the left stapes a fracture of the posterior crus of the right es, with callus formation. Reissner's mem- ne was torn and floating free throughout the hlea, and the organ of Corti was markedly phic. The spiral ganglion cells were reduced umber.

the third patient, a 46 year old welder, Reiss- s membrane was gone from both cochleas, tectorial membrane was absent, the organ of ti was not normal and the spiral ganglion were reduced in number. (Comment: Pre- ably, some of these lesions were the result of ssive acoustic stimulation, but until more of studies have been made one cannot be cer- that all of these lesions were due to acoustic ma.)

hamberlain [41] made audiograms on 4 boiler- kers just before, just after and at intervals r exposure to the noise of their trade, with following results: Patient 1 showed a slight porary loss at tone frequencies from 64 ough 1024 due to fatigue, with no further loss r six months of the same exposure. Patient owed a marked permanent loss for the higher quencies, probably of long standing. A slight itional loss was caused by noise, with essen- y complete recovery to the previous level in fifteen hours. Patient 3 showed a sig- cant loss after noise for all frequencies above except 8192 in the right ear. Recovery for s above 2048 had not taken place in fifteen rs, but in six months there was complete very except for a slight loss at the three est tones. Patient 4, with the same exposure atient 3, showed a much greater loss for all except the three lowest tones, with complete recovery in the right ear after fifteen hours; after six months the left ear had not recovered the hearing for the tones above 1024.

The author concludes that while the power of recovery from acoustic trauma is remarkable, it does not always take place within fifteen hours of rest from the noise. This suggests that adequate periods of rest between periods of exposure to noise might prevent or delay permanent cochlear damage. There is definite individual susceptibility to the effect of noise.

Dickson [42] finds that there is marked individual variation in susceptibility to aviation noise. Experimentally the maximum audiometric dip from excessive acoustic stimulation occurs an octave above a pure fatiguing tone. In airplane noise the notes of low frequency are those of greatest intensity, and yet the typical loss is at 4096. The explanation is not clear but may be that with a complex noise the damage is inflicted on the most susceptible part of the cochlea; or there may be sudden instantaneous peaks of intensity not recorded by the instruments at one's disposal, and these may be at 2048, causing the drop at 4096; or the external ear may act as a resonator intensifying certain frequencies of a complex tone.

The best protection against deafness from aviation noise is the wearing of a flying helmet with earphones attached. Other appliances, such as ear plugs, afford a certain protection but reduce intelligibility of speech.

Shilling and Everley [43] carried out a series of studies of hearing among submarine personnel to determine the losses due to exposure to diesel engine noise, exposure to radio noise and exposure to noise of gunfire.

After one hour's exposure to diesel engine noise, which averages 126 decibels of sound intensity, there was an average loss of 10.4 decibels; about five hours was required for hearing to return to normal. This loss involved all tones from 64 to 8192, being 10 decibels or less for the tones under 1024 and between 11 and 17 decibels loss for the tones from 1024 to 8192.

More than one hour's exposure caused a comparatively slight increase in the loss, but the recovery time was greatly lengthened, so that after two to four hours' exposure recovery was not complete even after twenty hours.

Radio men were found to have a hearing loss that varied with the length of exposure. It was

Wolff, D.: Microscopic Examination of Human yrinth from Patients Exposed to Loud Noises, h. Otolaryng. **36**:843 (Dec.) 1942.

Chamberlain, D.: Occupational Deafness: Audio- ic Observations on Aural Fatigue and Recovery, h. Otolaryng. **35**:595 (April) 1942.

42. Dickson, E. D. D.: Aviation Noise Deafness and Its Prevention, J. Laryng. & Otol. **57**:8, 1942.

43. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel: I., U. S. Nav. M. Bull. **40**:27, 396 and 938, 1942.

greatest for the tones of 4096 and 8192 cycles, as follows:

	Loss in Decibels at 4096	8192
After 30.3 months' exposure.....	10.5	15.0
After 85.3 months' exposure.....	23.3	23.5
After 148.2 months' exposure.....	28.1	31.8

The loss of hearing in these men was probably due to repeated acoustic trauma, such as "blasting" when nearby stations come in with great power while the radio man is listening to a distant station. The authors suggest that radio equipment be constructed so as to eliminate acoustic shock.

Hearing loss due to the noise of gunfire was found to affect the higher frequencies predominately, the loss for 2048 cycles being 19 decibels, that for 4096 cycles 33.4 decibels and that for 8192 cycles 35 decibels. The prevention of such deafness is best accomplished by properly applying cotton plugs or simply by placing the fingers over the external auditory canals and opening the mouth when an explosion is imminent. "Ear defenders" are uncomfortable after long wearing. (Comment: Otologists have become thoroughly aware of the dangers of deafness from acoustic trauma in modern mechanized warfare. Effective methods of prevention have not yet been worked out and generally adopted.)

Vetter [44] tested the hearing of 96 men in a motorized Swiss infantry gun unit which had been on active duty with frequent gunnery practice since mobilization nearly two years before. Only 15 had hearing defects or aural symptoms. Of these, 2 had no further examination, and only 1 of the other 13 had inner ear deafness with a sharp drop in the audiogram between 2,000 and 8,000 vibrations that could be ascribed to acoustic trauma. The wearing of cotton ear plugs during gunnery practice was routine.

The author concludes that artillerymen should always wear cotton ear plugs and should open their mouths during gunnery. With these precautions the likelihood of acoustic trauma in practice maneuvers is slight. The more frequent and prolonged shooting of active warfare, however, would probably gradually lead to inner ear deafness.

Weersma [45] found that persons with impaired hearing for low tones showed less than the normal hearing loss from noise, whereas with loss of hearing for high tones as in presbyacusis there is more than normal hearing loss from noise. (Comment: Loss of hearing for low tones is generally a conduction type of defect where the cochlea protected from acoustic trauma. In nerve deafness the nerve of hearing, already damaged, evidently more susceptible than normal to further damage from acoustic trauma.)

Allergic Nerve Deafness.—Taylor [46] notes that the administration of foreign serum has long been known to affect the peripheral nerves in occasional cases. The exact process of the neuritis is not known, but it is assumed to be an allergic perineural edema producing ischemia of the nerve. The average interval of time between the injection and the serum disease is seven days and with the neuritis there are usually fever, urticaria, headaches, nausea and vomiting and enlargement of lymph nodes.

Taylor describes a case of bilateral deafness of high degree with intense tinnitus coming on hours after the onset of a generalized anaphylactic reaction which developed five days after the patient received 5,000 units of tetanus antitoxin. There was no history of previous allergy but the patient had received tetanus antitoxin seven years previously. The perception deafness has continued without improvement.

Mao [47] reasoned that congenital deafness might be the result of repeated biologic trauma on the undeveloped cells of the fetal cochlea from absorption into the fetal circulation from the maternal circulation of antibodies and sensitizing agents in allergic mothers. Since allergy is regarded as definitely hereditary, the author made cutaneous tests on 535 pupils in the Pennsylvania School for the Deaf with the common allergens and found that 28.6 per cent of the congenitally deaf ones and 28.5 per cent of the ones with acquired deafness gave positive reactions. Since less than 1 per cent of normal children are allergic, the author concludes that while the occurrence of allergy is by no means selective as far as the two groups of deaf patients are concerned, it is definitely selective with regard to deaf persons and persons with normal hearing. He believes it reasonable to assume that allergy and deafness in combination have a hereditary background. (Comment: The author makes the common mistake of diagnosing allergy from the results of tests with allergens. These tests should be regarded as an aid but not as the means of diagnosis of allergy. The proof of allergy depends on the therapeutic test: relief from the symptoms on removing [or treating

44. Vetter: Otologic Studies of a Motorized Infantry Gun Unit, Schweiz. med. Wchnschr. **72**:1127, 1942.

45. Weersma, P.: The Effect of Noise on the Hearing of Normal and Hard of Hearing Individuals, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:402, 1941.

46. Taylor, H. M.: Neurologic Complications of Serum Sickness with Special Reference to the Eye, Laryngoscope **52**:923, 1942.

47. Mao, C.: Allergy as a Contributing Factor in Congenital Deafness, Arch. Otolaryng. **35**:582 (April 1942.

h] a specific substance, recurrence of the nptoms on exposure to the substance. Many n tests are positive without clinical allergy, some are negative in the presence of definite rgy.)

Congenital Nerve Deafness.—Hughson, Ciocco Palmer [48] classify deafness in children as mbryonic" when it is not associated with philis in the mother or with trauma in utero at birth or when the loss became manifest after th. The term "nonembryonic" is used when h association can be demonstrated. If the ld ever used articulated speech or acquired a abulary without special training, the deafness s classed as nonembryonic.

Of 307 children in the Pennsylvania School for Deaf, 35.8 per cent had embryonic deafness. afness was found to be more prevalent among relatives of these children than among those children with nonembryonic deafness. The idence seems to point definitely to the inheri- ce of embryonic deafness. If both parents ve embryonic deafness, all the children will be af; if one parent has embryonic deafness, one- lf or more of the children will be deaf, depend- g on whether the other parent is a carrier or is e from the gene; if neither parent is deaf but e or both are carriers, either none or 25 per nt of the children will be deaf. The presence embryonic deafness among the brothers or ters arouses the suspicion that a normal-hear- g child is a carrier. (Comment: The preven- n of congenital inherited or, as the authors call embryonic, deafness probably requires the plication of some sort of eugenic laws similar those in force in Germany. Accurate and liable statistics as to the operation and results those laws should be of great value if ever ailable.)

Nash [49] classified 218 pupils in the Rochester chool of the Deaf according to their hearing ss. Group I with a hearing loss in the better r of less than 40 decibels contained 3 pupils. roup II with a hearing loss of 40 to 60 decibels the better ear contained 16 pupils. Group III ith a loss of 60 to 80 decibels contained 56 upils. Group IV with a loss of 80 to 100 ecibels contained 143 pupils. In 5 there was no erception of sound in either ear.

Of the total 218 pupils, 36 per cent were efinitely benefited educationally by electrical earing aids, most of these being in the first three groups, but even in group IV the majority seemed to be helped in some way by the use of hearing aids. (Comment: While hearing aids can be of some help when the profoundly nerve-deafened child is being taught to speak, there is the possibility that exposure of these already damaged ears to excessively loud noises will cause further deterioration. If the child finds the hearing aid uncomfortable or painfully loud, damage to the nerve will result; if he does not find it uncomfortable or painful, the aid at that level of loudness is probably safe.)

Nerve Deafness of Unknown Cause.—Wever [50] made a detailed histologic study of the ears of 73 patients whose audiograms showed a tonal dip. The ears were classified into four groups according to the types of dip, as follows:

Group I was a control group of 16 ears with normal hearing for all tones up to 8192 cycles. The conditions of testing being taken into account, a fall of 20 decibels below the zero line is regarded as within normal limits.

Group II included 34 ears in which hearing was normal except for a depression of at least 15 decibels below the neighboring tones and at least 30 decibels below the zero line, the magnitude of the dip varying between 30 and 75 decibels. In 1 ear the dip was at 2048 and in 1 at 5793; in all the rest (32) the dip was at 4096. Even more striking was the fact that all the ears in this group but the ear with a dip at 5793 were from males.

Group III included 19 ears with hearing depressed 25 to 30 decibels for all tones, with a marked local dip and a partial return to normal above 4096. In 1 ear the dip was at 5793; in 18, at 4096. Two of these ears were from a woman; the rest were from males.

Group IV consisted of 4 ears in which the dip did not reach a point 30 decibels below the zero line and which were considered therefore to show a borderline condition.

In the histologic study of this material, each tenth section was examined and the condition of the organ of Corti, the nerve fibers, the external sulcus cells and the stria vascularis was recorded, with the following results:

In the control group (I) a moderate amount of pathologic change was present in ears in which hearing was within normal limits of acuity. Indeed, it was exceptional to find an ear that was histologically perfect. Perhaps the most common defect was atrophy of the nerve fibers at the lower basal end. Also common were scat-

48. Hughson, W.; Ciocco, A., and Palmer, C. E.: tudies on Pupils of the Pennsylvania School for the eaf: IV. Mechanism of Inheritance of Deafness, Arch. tolaryng. **35**:871 (June) 1942.

49. Nash, C. S.: Residual Hearing, Laryngoscope **2**:577, 1942.

50. Wever, E. G.: The Problem of the Tonal Dip, Laryngoscope **52**:169, 1942.

tered regions of moderate atrophy of the stria vascularis.

In group II there was in general a greater amount of pathologic change than in the control group. In more than half the ears partial atrophy of nerve fibers extended over the entire lower basal half-turn and in a few ears even farther. As a rule, the atrophy was greatest at the basal end and decreased rapidly as the spiral proceeded apically. Pathologic change of the organ of Corti was likewise somewhat more common than in the control group, yet the atrophy was only slight, and for the most part was confined to the extreme basal end. It is doubtful that these observations can fully account for the specific losses of acuity, for they were no more common with the larger dips then with the smaller. Atrophy of the external sulcus cells was much more common than in the control group, yet in degree and extent it was no greater than the worst atrophy in the control group. No particular region of the cochlea was found which could certainly be correlated with acuity for 4096 cycles; the atrophy that was present was nearly always at the basal end, except that of the stria vascularis, which was more scattered.

In group III the amount of damage was slightly greater on the whole than that in group II, yet its amount was hardly commensurate with the degree of auditory impairment except in 2 or 3 instances.

In group IV the histologic picture showed no significant abnormality.

The author concludes that the tonal dip is not correlated in any convincing way with the histologic condition of the cochlea. Generally speaking, ears for which hearing tests demonstrate dips, especially if these are deep and wide, show somewhat more atrophy than normal ears, yet there are many exceptions. No specific areas can be located whose condition varies in any consistent manner with the acuity for 4096 cycles. The areas of atrophy that are present are usually at the basal end, except for those of the stria, which are more widely scattered. This is in contrast with the work of Crowe, Guild and Polvogt, who correlated losses of acuity for high tones with areas of atrophy in the basal region of the cochlea.

Wever believes that the cause of the tonal dip is most often acoustic trauma. Since the lower basal portion of the organ of Corti is the region of greatest acuity for the ear as a whole, it is possible that the high degree of sensitivity of this region makes it particularly fragile and susceptible to the damaging effects of acoustic trauma.

Carrasquillo [51] reports a case of Vogt-Koyanagi syndrome, a clinical entity characterized by severe spontaneous bilateral uveitis, white areas in the skin and falling of the hair, often associated with loss of hearing. The hearing loss occurs in about 50 per cent of the cases; it appears shortly after the onset and is accompanied by tinnitus and hyperesthesia to noise. The deafness is transitory in many cases.

In the instance reported by the author there was bilateral high tone nerve deafness, the audiometric curve showing a fairly uniform descent in both ears, beginning at 25 decibels loss for the 128 frequency down to 100 decibels loss for 11584. The cause of the syndrome is unknown. There is no known treatment, and the disease usually runs a protracted course ending in partial or complete blindness.

McGovern [52] states that retinitis pigmentosa is frequently associated with gradual progressive high tone nerve deafness, the reported incidence being 10.4 to 44 per cent. Hereditary abnormality and consanguinity are found in 40 per cent of the cases of retinitis pigmentosa.

The author reports a family of 8 sons and daughter in which 4 of the sons had retinitis pigmentosa with constricted fields and great restricted vision and had also nerve deafness with a gradually increasing defect for the higher frequencies. The mother and the father were first cousins.

Treatment of Nerve Deafness.—Lurie [53] lists the possible causes of nerve deafness as follows: destruction of the central nerve pathways, as by hemorrhage, tumors or arteriosclerosis; degeneration of the cochlear nerve from meningitis, syphilis, acoustic neuroma and toxic neuritis; degeneration of the hair cells from alcohol, tobacco, quinine and salicylic acid, pneumonia, influenza, mumps, syphilis and meningitis.

Lurie emphasizes that one must remember that in such cases of nerve deafness no cure can be promised or expected from vitamins, estrogenic substances or other therapeutic agents. The only patients with nerve deafness whom he has helped with thyroid had definite hypothyroidism with loss of hearing due to slow cerebration rather than to loss of cells in the sense organ.

There is a group of patients who inherit a tendency to acquire nerve deafness, and the history

51. Carrasquillo, H. F.: Uveitis with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **28**:385 (Sept.) 1942.

52. McGovern, F. H.: Association of Nerve Deafness and Retinitis Pigmentosa, Arch. Otolaryng. **36**:82 (Dec.) 1942.

53. Lurie, M. H.: Medical Progress: Otology: The Treatment of Deafness in the Light of Recent Animal Experimentation, New England J. Med. **226**:886, 1942.

icates that members of their families have tained the same progressive nerve deafness ning on between puberty and 45. Certain mals show this same tendency. Nothing can done for these patients with inherited acquired rve deafness.

As to treatment for nerve deafness, Lurie ades raising the general physical well-being of patient to as high a level as possible. Vitains and other drugs should be used to bring s about. Foci of infection should be removed. oxic substances, such as tobacco, alcohol and ugs, should be completely eliminated. The oustical engineer must reduce as far as posle any noise that may cause the organ of Corti degenerate. (Comment: This is an excellent d sound approach to the treatment of nerve afness. To the etiologic factors should be ded primary labyrinthine otosclerosis, a conion that cannot be diagnosed with certainty ring life but that can be suspected in cases of rve deafness if there is a family history of otolerosis or if there is a well marked Schwartze's gn [pinkish glow] from the promontory.)

Criswell [54] states that the prevention of deafss in aging persons depends on the removal of iologic factors causing deafness earlier in life, nce the deafness in old age is purely degenerave.

Fowler [55] calls vibratory tinnitus the actual unds resulting from: contractions of the intympanic muscles or the tubal muscles; aneusms, angiomas, bruits in the jugular bulb and her hemic murmurs; clicks and squeaks about e jaw joints; spasms of the facial and cervical uscles; bubbles in the eustachian tube; various spiratory and digestive motions. Vibratory nitus, if loud enough, can be heard by the xaminer, and its loudness is real, not illusory.

Nonvibratory tinnitus is not due to an actual und but originates by local or reflex biochemial irritation of the neural elements of the audiry apparatus. It is an illusion of sound. The atient may describe it as loud, but the measured udness may be only 5 to 10 decibels.

Fowler finds that the tone of a tinnitus correponds usually to the upper portion of a dip in he audiometric curve, indicating that the nonibratory tinnitus of a particular tone comes from ocal irritation of areas of the basilar membrane, whereas tinnitus with a diffused tonal quality is nore apt to be of central origin.

The following disorders are associated with nonvibratory tinnitus: Ménière's syndrome; acoustic trauma; intracranial sclerotic lesions; aneurysms; tumors of the brain; anoxia; changes in hydrogen ion concentration; allergic conditions and disturbances of water balance; toxic degenerative and regenerative biochemical irritations of the auditory nerves; effects of drugs, such as quinine, the salicylates, nicotine, arsenic, phosphorus, lead, carbon monoxide, carbon disulfide, mercury, morphine and some aniline dyes.

The treatment of tinnitus requires a careful study to determine its etiologic factors, contributing factors and prognosis. Removal of impacted cerumen and unblocking of the eustachian tubes is, of course, indicated. Allergies, hypotension or hypertension, pathologic involvement of the temporomandibular joint and abscesses or impaction of teeth should be corrected. The taking of quinine, salicylates, arsphenamine, nicotine, alcohol and caffeine should be discontinued. The general health should be improved. (Comment: Fowler is to be complimented for his studies on this most frequent, most annoying and most poorly understood symptom. When tinnitus is associated with an obvious reversible pathologic process such as impaction of cerumen, secretory otitis media or acute suppurative otitis media, removal of the cause can be expected to relieve the tinnitus. In some [but not all] cases of otosclerosis a successful fenestration removes or diminishes the tinnitus. But in the great majority of cases of tinnitus otologists are able neither to determine the etiologic factor nor to relieve the tinnitus by any local or general therapy in the present state of knowledge.)

Shambaugh and Jennes [56] stress the need for adequate controls in the assessment of any treatment for deafness, because of the psychic element in the response of deaf persons to any new therapy. A deafened person easily falls into the habit of inattention; stimulated by hope through a new treatment, he begins to pay more attention and therefore hears more, though his hearing remains unchanged to objective tests, for these always require his full attention. Audiometric measurements must be relied on, but caution must be observed here too, for either because of spontaneous fluctuations in the hearing from fatigue and other factors or because of the errors inherent in audiometric tests, there may be fluctuations in the audiometric hearing in any case of deafness without therapy. Finally, no conclusions can be drawn from the results of

54. Criswell, J. R.: Symposium on the Medical Problems of the Aging, Hahneman. Monthly **77**:236, 942.

55. Fowler, E. P.: The "Illusion of Loudness" of Tinnitus: Its Etiology and Treatment, Laryngoscope 2:275, 1942.

56. Shambaugh, G. E., Jr., and Jennes, M. L.: Therapy of Nerve Deafness and Tinnitus Aurium, Arch. Otolaryng. **35**:513 (April) 1942.

therapy in cases of deafness due to self-limited conditions, in which the tendency is toward recovery without treatment. It is particularly easy to overlook secretory otitis media, which may cause a hearing impairment simulating nerve deafness or which may be superimposing additional impairment on chronic deafness. Since secretory otitis media tends to clear up spontaneously and the functional tests suggest nerve deafness, it would be easy to conclude that the given form of therapy had resulted in improvement in a case of nerve deafness.

The authors, disappointed in the results of treating nerve deafness by the usual doses of vitamin B_1, gave daily intravenous injections of 50 mg. of thiamine hydrochloride over a period of two weeks to 5 patients with progressive nerve deafness of long standing. The hearing was tested audiometrically before and after the treatment, and not one of the patients experienced any subjective or objective improvement in hearing. In no case was there any relief from tinnitus.

From their experience and from a review of the literature the authors conclude that there is no satisfactory evidence in favor of the use of thiamine hydrochloride for the alleviation of deafness and tinnitus. It seems to the authors that since well developed deficiency states such as beriberi, scurvy and pellagra are not known to be associated with loss of hearing, it is unlikely that subclinical deficiencies produce nerve deafness.

Neostigmine was employed for the relief of tinnitus by four authors during 1942. Schluederberg [57] gave semiweekly injections of a solution of neostigmine methlysulfate, 1 cc, of a 1:2,000 solution, with ingestion of 15 mg. of neostigmine bromide three times daily on the days when the injections were not given. Of 20 patients with perceptive deafness, 2 showed hearing improvement, 2 relief from tinnitus, 3 diminution of tinnitus and 4 a doubtful result. Of 8 patients with conductive deafness, 1 showed hearing improvement, 2 lessening of tinnitus and 1 doubtful improvement. Of 10 patients with mixed types of deafness, 2 showed hearing improvement, 3 lessening of tinnitus and 2 doubtful improvement.

At least one audiogram was taken before treatment began, and others were taken at two week intervals during treatment. The hearing was considered improved when the decibel losses for the frequencies 512, 1024 and 2048 were reduced 15 decibels or more. In every case of improvement in hearing, tinnitus too was improved, and it is quite possible that the improvement in hearing was the result of the lessening of tinnitus.

The author concludes that neostigmine merits further consideration before it is rejected for the treatment of deafness and tinnitus. (Comment: This was a carefully made study of cases, but a series of several, preferably four, audiograms made before the start of treatment, to establish the pretreatment level of hearing, would have been better than the single audiogram. We have found that the second and third pretreatment audiograms will frequently show an improvement of 5 to 10 decibels over the first audiogram simply as a result of practice. The value of the author's study would have been greatly enhanced by the use of controls who received placebos by injection and by mouth.)

Ysander [58] injected a solution of neostigmine methylsulfate, 1 cc. of the 1:2,000 solution, every three or four days, followed by the taking of neostigmine bromide tablets daily by mouth for several weeks, for tinnitus in 27 patients, with complete relief in 2, improvement in 13, doubtful result in 10 and no result in 2.

Taterka [59] injected 1 cc. of a 1:2,000 solution of neostigmine methylsulfate two times a week with 3 neostigmine bromide tablets being taken by mouth every other day. The number of injections varied from 6 to 30. Of 21 patients treated, 12 claimed improvement in their tinnitus. Many commented about the improvement in hearing, but no audiometric studies were made. The decrease in tinnitus probably explains the patients' believing that they heard more acutely.

Judge [60] injected 1 cc. of a 1:2,000 solution of neostigmine methylsulfate three times weekly or a 15 mg. neostigmine bromide tablet was taken by mouth twice daily. Of 14 patients with tinnitus, 5 obtained complete relief, 4 had marked improvement and 3 some benefit. Audiograms of 16 patients taken before and after conclusion of the treatment showed a significant improvement of hearing in 3.

The author concludes that neostigmine therapy is of definite value in the treatment of tinnitus aurium, and its use in the treatment of deafness should not be abandoned in view of the paucity of therapeutic agents for the treatment of impaired hearing. (Comment: These four articles on the use of neostigmine for the treatment

57. Schluederberg, H.: The Treatment of Deafness and Tinnitus Aurium with Prostigmine, Ann. Otol., Rhin. & Laryng. **51**:526, 1942.

58. Ysander, F.: Prostigmine in Tinnitus Aurium, Schweiz. med. Wchnschr. **72**:781, 1942.

59. Taterka, H. D.: Preliminary Study of Effects of Prostigmine on Tinnitus, Laryngoscope **52**:299, 1942.

60. Judge, A. F.: Prostigmine in the Treatment of Chronic Deafness and Tinnitus Aurium, Mil. Surgeon **90**:177, 1942.

nitus came to conclusions contrary to those ached by Ersner, Rush and Myers a year fore and by Hansen, Campbell and Schlueder- g two years before, although the dosage used s identical and the types of deafness treated ere similar. Our own experience coincides th that of the authors of these earlier studies, mely, that neostigmine has been of no definite nefit in cases of deafness and tinnitus.)

Nerve Deafness of Central Origin.—Paredero Bosque [61] reports on 2 patients, one with a und in the left temporal region and one with wound in the right temporal region, who had nerve type of hearing impairment in the ear the opposite side. In each case there was no evious disease of the auditory apparatus; in ch case the functional hearing tests were con- nt, excluding hysterical deafness; in each case other etiologic cause for deafness of the nerve e could be found. The author concludes that fracture of the internal lamina of the skull er the primary and secondary cerebral con- utions caused compression where the large tical fibers of the central acoustic fasciculus minate, resulting in central deafness. (Com- nt: Since surgical removal of one cerebral misphere does not result in deafness in the posite ear, owing apparently to the fact that h ear is represented in both the right and left cerebral cortex, it seems unlikely that deafness of these patients was of central gin and much more likely that the skull trauma s accompanied by labyrinthine hemorrhage in opposite ear.)

De Crinis [62] reports a case of sudden vertigo, in in the right ear and vomiting, with paralysis the right side of the face. A whisper was heard rmally in the left ear, but not at the ear on the ht, whereas pure tones were heard equally ll in both ears. The spinal fluid showed a rked increase in lymphocytes, from which the thor diagnosed meningoencephalitis with in- vement of the midbrain resulting in the deaf- ss for speech with retained hearing for pure es. The author cites 4 similar cases reported Buttich in 1940. In 1 case the deafness was result of an inflammatory disease; in 1, of a mor of the brain, and in 2 cases, of cerebral uma.

If the lesion lies between the labyrinth and the chlear nucleus in the brain stem, hearing for eech and for pure tones is equally affected. If the lesion involves the midbrain, the hearing for pure tones is retained but that for speech is affected, according to the author. (Comment: This is an interesting contribution to the meager knowledge of deafness of central origin.)

Falkenberg [63] states that a lesion of the lateral lemniscus of one side leads to bilateral hearing loss, more marked in the opposite ear. The hearing tests show the Rinne test positive, the Weber test heard in the better ear, bone conduction markedly shortened, the limits of hearing for the upper and lower tones shortened and the hearing for the lower tones more marked, without tone islands. There is also marked impairment of hearing for speech.

According to the author, deafness caused by damage of the lateral lemniscus cannot be diagnosed from the hearing tests alone but can be suspected from the course of the disease and from the other neurologic symptoms. However, the hearing tests may help in localizing the process.

Mollison [64] believes that deafness in which the patient can hear single words but cannot grasp whole sentences is attributable to sluggishness of the central perceiving apparatus, that is, a central lag. Many of the patients with this type of deafness show a drop at 4096 in the audiogram. (Comment: It is characteristic of the patient with high tone nerve deafness to hear without perceiving; that is, the high tone loss with loss of hearing for the consonants prevents him from understanding, whereas he hears the vowels without difficulty. Perhaps some of Mollison's cases with the dip at 4096 could be explained on this basis.)

Ménière's Disease.—Lindsay [65] describes the histologic changes in the ear of a man of 67 with a history of noises and advanced deafness in both ears. The right labyrinth showed extreme dilatation of the ductus cochlearis at the expense of the scala vestibuli, with extensive dilatation of the saccule, as well as of the utricle, which had herniated into the small ends of the semicircular canals and into the ampullas of the horizontal and posterior vertical canals so as to displace the wall of the ampulla opposite the cupola and the crista. Corti's organ and the nerve elements were well preserved except for the hair cells, which showed extensive degeneration. The left labyrinth showed similar changes but with less extensive dilatation and herniations.

61. Paredero del Bosque, J.: Deafness of Central gin Due to Traumatic Lesion: Two Cases, Medicina, drid **10**:52, 1942.

62. de Crinis, M.: Deafness for Speech with Retained aring for Pure Tones, Arch. f. Ohren-, Nasen- u. hlkopf. **149**:183, 1941.

63. Falkenberg, K.: Mesencephalic Deafness, Arch. f. Ohren-, Nasen- u. Kehlkopf. **149**:187, 1941.

64. Mollison, W. M.: Deafness Due to Central Lag, J. Laryng. & Otol. **57**:420, 1942.

65. Lindsay, J. R.: Labyrinthine Dropsy and Ménière's Disease, Arch. Otolaryng. **35**:853 (June) 1942.

These findings are similar to those previously described in cases of Ménière's disease. The author believes that the attacks of vertigo can be explained on the basis that the utricle herniating into the canals produced distortion of the walls of the ampullas. The loss of hearing he explains also on a physical or a mechanical basis: The distortion of Reissner's membrane and the resulting disproportion of the scala media and the scala vestibuli suggest interference with sound transmission in the column of fluid in the cochlea as the cause of disturbed function.

The histologic picture explains the favorable effect of certain types of treatment, such as the limitation of the intake of fluids, the salt-free diet and the use of ammonium chloride or potassium salts.

Lindsay [66] believes that the accumulating evidence of the past few years indicates that a definite pathologic alteration consisting of dilatation of the endolymphatic spaces is the basis of recurring attacks of vertigo with fluctuating auditory disturbances. Lindsay believes that the name Ménière's disease can be applied to the clinical entity, and hydrops of the labyrinth to the histologic changes.

A total of 6 proved cases of Ménière's disease have been examined histologically, and 5 more cases of deafness with the same dilatation of the endolymphatic spaces. Since Ménière's disease occurs in people with low blood pressure, vasomotor instability, headaches and gastrointestinal disturbances, it seems likely that the increase in the endolymph is the result of vasomotor disturbances in the stria vascularis or the aqueductus endolymphaticus. Although a few cases have been reported in which attacks of vertigo and impaired hearing occurred in connection with allergic attacks, the author believes that one is not justified in regarding Ménière's disease as an expression of allergy.

The most successful medical treatment has been on the basis of attempting to control the fluid balance by limitation of the intake of salt and control of the intake of fluids and by administration of ammonium chloride or 3 to 4 teaspoons a day of 25 per cent potassium chloride solution, with adequate rest and mild sedation. Histamine therapy has not met with general lasting success, and inflation of the eustachian tubes has been quite unsuccessful in the author's experience. In cases resistant to medical treatment operation on one or more semicircular canals stops the vertigo, while preserving the hearing, and is less dangerous and incapacitating than intracranial section of the vestibular nerve.

66. Lindsay, J. R.: Ménière's Disease, Minnesota Med. **25**:778, 1942.

Merica [67] believes that obstruction of the eustachian tubes is a frequent cause of vertigo. During the past few years he has seen 135 patients with vertigo, nausea, poor appetite, "bilious attacks," constipation, nervousness and many other symptoms completely relieved by reestablishment of the patency of the eustachian tubes. This was accomplished by catheterizing the tube, passing a bougie and then inflating the tube. In some cases symptoms present for months or years were removed by a single inflation; in others recurrent attacks required repeated inflations. (Comment: Contrary to the author, our experience has been that patients with vertigo rarely have tubual occlusion and that patients with tubal occlusion rarely have vertigo. We agree with Lindsay that inflation of the eustachian tubes has been quite unsuccessful in the treatment of Ménière's disease.)

Asenjo and Rococo [68] distinguish four different types of Ménière's disease: the true Ménière's disease, exceedingly rare, with exudate in the semicircular canals at autopsy; the symptomatic Ménière's disease, in which the vertigo is a symptom of some primary condition, such as a tumor; pseudo Ménière's disease, in which there is vertigo without other symptoms of Ménière's syndrome, with vertigo, deafness and tinnitus.

Because of the spontaneous remissions in cases of Ménière's syndrome, the authors recommend four to six months of medical treatment, with surgical treatment only when medical treatment gives no results. Medical treatment consists of Furstenberg's regimen, ammonium chloride, potassium chloride, vitamin B_1 (injected) and a preparation of phosphorus. In a series of 21 patients showing Ménière's syndrome the authors obtained good results in 12 of 18 who received medical treatment and in all 3 of those who were operated on.

C. I. Johnson [69] classifies vertigo into central and peripheral, the former being generally non-episodic, nonsystemized and of longer duration. Of particular interest is the Ménière's syndrome of unknown cause in which all possible medical, neurologic and otologic causes have been ruled out. The author discusses the various medical treatments of this group without expressing an opinion as to the best.

67. Merica, F. W.: Vertigo Due to Obstruction of the Eustachian Tubes, J. A. M. A. **118**:1282 (April 11) 1942.

68. Asenjo, A., and Riesco, J. S.: Ménière's Disease, Rev. de otorrinolaryng. **2**:83, 1942.

69. Johnson, C. I.: Ménière's Disease, Ann. Otol. Rhin. & Laryng. **51**:676, 1942.

Cawthorne, Fitzgerald and Hallpike [70] in a ost interesting and original contribution report e results of a new type of caloric test in a series 50 cases of Ménière's disease. The authors lieve that the maximum delicacy and precision the caloric test are attained by using 2 pints ,000 cc.) of water at 30 C. for the cold caloric d at 44 C. for the hot caloric test, run into e ear in a fairly heavy stream in about forty conds.

The nystagmus provoked by this test in a nor- al person lasts for about one and a half to two d a half minutes, is easy to observe and is rely accompanied by nausea or severe vertigo. artels glasses are not used, as they seem to ffuse the end points. The patient lies on a uch with his head raised about 30 degrees to ing the horizontal canals to the vertical plane. e fixes his gaze on a mark on the ceiling, and e duration of nystagmus from the onset of rigation to the end of the response while he oks straight ahead is recorded. Since each ear tested with both hot and cold water, 4 tests e required, with a five minute rest between.

In 50 normal subjects the duration of the ystagmus after the cold stimulus varied between e and two-tenths and two and four-tenths inutes; in 80 per cent it was between one and ree-tenths and two and one-tenth minutes. The ystagmus after the hot stimulus was slightly orter, usually by five to fifteen seconds, than at after the cold stimulus, in 80 per cent of ses; it was equal to the latter in 10 per cent d was longer than the cold reaction in 10 per nt. Between the two ears of the normal group e reactions differed in duration by less than ve seconds.

Abnormal reactions took three forms: 1. A irectional preponderance such that nystagmus ward the right or the left side, whether in- uced by a hot or a cold stimulus in either ear, owed greater duration than nystagmus induced ward the opposite side. 2. A one-sided pre- onderance such that the reactions to both hot nd cold water were of longer duration with one ar than with the other. 3. A combination of e first two types.

The authors regard the utricle as the chief ource of tonic impulses from the human laby- inth, its elimination on one side leading to direc- onal preponderance of nystagmus. In a patient ith a unilateral labyrinthine lesion directional reponderance is pathognomonic of utricular paresis. However, directional preponderance can also come from a central vestibular disturbance and was present in every one of 10 cases of tumor of a temporal lobe.

One-sided preponderance means paresis of the horizontal canal alone, with an intact utricle.

A combination of the two types means paresis of both the utricle and the horizontal canal.

Of 50 patients with Ménière's disease tested, only 3 showed normal caloric reactions. In 3 a directional preponderance to the left indicated right utricular paresis, while in 7 a directional preponderance to the right indicated left utricular paresis. In 15 there was paresis of the left horizontal canal; in 14, paresis of the right canal, while in 8 there was paresis of both the utricle and the canal.

The authors believe that Ménière's disease is a characteristic clinical picture due to obstructive distention of the endolymphatic system. In all of the author's 50 patients there was loss of hearing to the whisper test, and in 86 per cent hearing for the whisper was reduced from a normal of 30 feet to 20 feet or less in both ears. The deafness was nearly always of the inner ear type. Neither pathologic involvement of the eustachian tube nor tympanic disease nor foci of infection appeared to play an important etiologic role. In most cases the ear with the worse hearing showed the vestibular lesion, but in 5 cases the vestibular disturbance as revealed by the caloric test was in the ear with the better hearing. (Comment: The latter observation is of particular interest when one comes to surgical intervention for the relief of vertigo and is another reason for operating only as a last resort.)

Wright [71] describes a simplified technic for injecting alcohol into the labyrinth for Ménière's disease. A towel with a hole in it is placed over the ear, and he uses the trigger release syringe described by him in 1938, which was designed to permit the maximum degree of tactile sense when piercing the footplate of the stapes. A loosely filled horseshoe-shaped sandbag steadies the head, and the alcohol is tinted with methylene blue.

Apart from the 2 early cases in which facial palsy was induced, in one case permanently, in the other temporarily, no untoward incidents have occurred, and the author is satisfied that if the operator takes the precaution of observing for facial movements before making the injection, and does not inject more than 2 minims (0.12 cc.), no harm to the facial nerve should result.

However, he feels that the simplicity of the operation may lead to its being employed when

70. Cawthorne, T. E.; Fitzgerald, G., and Hallpike, S.: Studies in Human Vestibular Functions: III. Observations on the Clinical Features of "Ménière's" Disease, with Especial Reference to the Results of the Caloric Tests, Brain **65**:161, 1942.

71. Wright, A. J.: Ménière's Disease: Alcohol Injection of the Labyrinth, J. Laryng. & Otol. **57**:120, 1942.

it is not indicated. Destruction of the labyrinth when complete does abolish the vertigo of Ménière's disease, provided the one and only responsible labyrinth has been dealt with; on the other hand, it inevitably destroys what hearing may remain, and, most important of all, it does nothing to safeguard the other side. He believes that the correct treatment of Ménière's disease is to use every effort to cure the responsible focus of infection before resorting to labyrinthine destruction. (Comment: Surgical opening of the horizontal semicircular canal to destroy the ampulla of this canal may prove to be preferable to injection of alcohol, since it may preserve the hearing.)

Atkinson [72] calls attention to the fact that less than one fourth of all patients with Ménière's disease are histamine sensitive as determined by the intracutaneous test described by him. Only this small group can be treated effectively by gradual desensitization to histamine. The much larger group which gives a normal cutaneous reaction to histamine may experience temporary improvement from the vasodilator effect of this drug but cannot be treated satisfactorily with histamine for any length of time. In this larger group the disease is due to vasoconstriction and responds to nicotinic acid and other vasodilators. The author believes that histamine will not relieve all or even most patients of Ménière's syndrome and that it should be reserved for the histamine-sensitive ones as determined by the cutaneous test. (Comment: Our experience with histamine in the treatment of Ménière's disease has been disappointing. Perhaps it would have been better had we treated only the histamine-sensitive patients as urged by Atkinson.)

Shea [73] regards Ménière's disease as the result of a production of toxins by the liver, the gallbladder and the colon and believes that histamine acts by relieving this autointoxication. As a test he injects 0.5 cc of histamine (2.75 mg. in 5 cc.) hypodermically. If the symptoms produced are those of which the patient complains, the prognosis for histamine therapy is favorable. For mild and moderate intoxication 0.5 cc. of the solution just described is injected two or three times a week; to prevent future attacks the dose may be increased. For acute and severe vertigo 2.75 mg. of histamine diphosphate dissolved in 250 cc. of isotonic solution of sodium chloride is given intravenously, 1 drop per second, as advised by Horton. The author does not present his results but claims that this treatment is successful in the majority of cases. (Comment: Because Ménière's disease is a chron disease running an irregular course, wi periods of exacerbation alternating with perio of remission, and the patient comes to the doct usually only during the height of an exacerbati there is a tendency toward improvement irrespe tive of the therapy used. Great caution mu therefore be exercised in interpreting the resul of therapy in this condition.)

Weiss [74] discusses the treatment of vertigo an syncope from the standpoint of the internist. H emphasizes the need for distinguishing betwee vertigo and syncope, two symptoms which a both referred to by the patient as "dizziness" b which are unrelated in their genesis and trea ment.

In vertigo the patient has the experience rotation, usually with ataxia. This sensation ca be reproduced by whirling the patient. Vertig is always due to a disturbance of the vestibul apparatus, peripheral or central. It can b caused by disease of the labyrinth, concussion the brain or lesions of the fourth ventricle.

In the treatment of vertigo the first step is th elicitation of the specific cause. Elimination foci of infection is usually of no avail or at be of only secondary importance. There are n drugs with a specific effect on vertigo. Fo mild vertigo the taking of bromides 0.75 to Gm. (10 to 15 grains) every four to six hour can be helpful. The multiplicity of the thera peutic agents advocated for Ménière's disea raises a suspicion concerning their true efficac Therapeutic optimism, lack of adequate control irregular occurrence of the attacks and, most o all, lack of appreciation of the fact that in man instances Ménière's syndrome is transient an of functional origin explain the short life of thes various therapeutic claims.

Weiss lists the therapeutic measures currentl in use for Ménière's disease, including Furste berg's diet, the Mygind-Dederding limitation o the intake of fluid, the Talbot-Brown potassium chloride therapy, the Harris-Moore treatmen with nicotinic acid and thiamine, and the Shel don-Horton histamine therapy. He finds it diffi cult to understand the rationale of the intravenou injection of histamine advised by the latter, sinc the known bodily effect of histamine is of shor persistence. He regards Atkinson's theory tha a vasomotor disturbance, either allergic or vaso spastic causes Ménière's syndrome, as based o meager evidence.

In the treatment of Ménière's syndrome Weis advises avoidance of infection and of physiologi

72. Atkinson, M.: Histamine in the Treatment of Ménière's Syndrome, J. A. M. A. **119**:4 (May 2) 1942.

73. Shea, J. J.: The Office Treatment of Vertigo (Histamine Therapy), Laryngoscope **52**:363, 1942.

74. Weiss, S.: The Treatment of Vertigo and Syn cope, J. A. M. A. **118**:529 (Feb. 14) 1942.

d psychic stress. A diet low in sodium or the
inistration of potassium chloride may be
ed, but their value has not yet been estab-
hed. If after one or two years of carefully
nducted medical treatment improvement does
t occur, and if the attacks are severe and fre-
ent, surgical intervention may be considered,
t it is indicated in but a small, selected group.

Syncope, with dizziness, giddiness, headaches,
akness, dimness of vision and "blackout," is as
rule the result of sudden and transient cerebral
hemia. It usually is due to a derangement in
e distribution of the blood volume but may be
e result of hemorrhage. Although recovery
om syncope is the rule, it can be fatal, par-
ularly if the upright position is maintained.

The treatment of syncope depends on discovering and treating the underlying etiologic factors responsible for the cerebral ischemia. These factors are: psychogenic factors; structural lesions of the central nervous system; gravity, chemical factors, including anoxia; hemorrhage; cardiac reflexes (vagal inhibition); heart block; paroxysmal auricular or ventricular tachycardia; hypoglycemia; effects of local anesthetics; hypotension. (Comment: This is an excellent and valuable discussion of dizziness due to cerebral ischemia as differentiated from vertigo.)

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Abstracts from Current Literature

Ear

SIMULATION OF DEAFNESS. WILLIAM E. GROVE, Ann. Otol., Rhin. & Laryng. **52**:573 (Sept.) 1943.

Grove points out that simulation of deafness may be a conscious or an unconscious act. The conscious act represents a definite attempt to defraud, while the unconscious act represents an abnormal state of mind. The otologist must be able to detect both, but the treatment of the latter lies with the psychologist. The paper deals particularly with conscious simulation and gives an excellent review of the various tests which may be employed in unmasking the malingerer.

M. V. MILLER, Philadelphia.

OTOSCLEROSIS. HANS BRUNNER, Laryngoscope **53**:736 (Nov.) 1943.

Brunner presents his experiences with otosclerosis inasmuch as they do not agree in some points with statements in recent textbooks. He presents 17 cases studied both clinically and pathologically and 1 case studied only clinically.

Otosclerosis and Sex.—It is usually felt that otosclerosis affects women more than men. Clinically this may be true, but pathologically more lesions are found in men than in women. Some of the men, however, had few, if any, symptoms clinically.

Deafness and Otosclerosis.—Twelve of the 17 patients noted showed diminished hearing. Of this group, 8 were women and 4 were men, showing the preponderance of women as far as deafness due to otosclerosis is concerned. The deafness may be due to (*a*) ankylosis of the stapes, (*b*) partial obliteration of the holes of the tractus spiralis forminosus and (*c*) a congenital malformation of the cochlea. Ankylosis of the stapes is of great importance as to the diminution of hearing in otosclerosis, but such diminution may occur with other lesions and without ankylosis of the stapes. One must remember, therefore, that otosclerosis does not always result in ankylosis of the stapes and that every stapedial ankylosis is not due to otosclerosis. Otosclerosis is not always bilateral. In 13 cases both temporal bones were examined, and in 4 of these the involvement was unilateral. In none were there clinical symptoms.

Tinnitus and Otosclerosis.—As to 5 of the 17 patients, the author does not know whether or not they suffered from tinnitus, but of the remainder (12) there were 11 without tinnitus, and only 1 in whom tinnitus was a predominant symptom. He concludes that neither ankylosis of the stapes nor atrophy of the cochlea necessarily produces tinnitus.

Pregnancy and Otosclerosis.—The author feels that in some cases otosclerosis is not affected by pregnancy and that in others the hearing diminishes rapidly without pregnancy. He presents several cases which tend to prove these statements. He feels that otosclerosis may not be influenced by the usual course of a pregnancy but rather, perhaps, by accessory features, such as endocrine, vasomotor or physical disturbances; if this is not true, perhaps there are several kinds otosclerosis, only some of which are influenced by usual pregnancy.

HITSCHLER, Philadelphia

MÉNIÈRE'S DISEASE: SURGICAL APPROACH TO LABYRINTH WITH DESTRUCTION OF THE UTRIC CASE REPORTS. HENRY M. GOODYEAR, Laryngoscope **53**:743 (Nov.) 1943.

The surgical approach to Ménière's disease is only when the disease does not respond to med treatment. Postmortem examination in cases of Ménière syndrome has shown great dilatation of the saccule the scala media. The author, therefore, felt that compression of the labyrinth would give relief.

Using a postauricular incision, he removed the b between the ampullas of the horizontal and super semicircular canals. The decompression was covered with a skin flap. This operation gave fair results. The author then decided to remove destroy the utricle. The approach was similar to just described and a small dental excavator was to destroy the utricle.

Three cases are presented with good results. Ho ever, in each case all hearing on the treated side destroyed. The author hopes in the future to limit destruction to the ampullas of the superior and ho zontal canals and possibly to include only the otoli membrane, with the hope that the hearing may retained.

HITSCHLER, Philadelphia

Larynx

NONSURGICAL ASPECTS OF TREATMENT OF AC LARYNGOTRACHEOBRONCHITIS. HARRY L. BA Ann. Otol., Rhin. & Laryng. **52**:608 (Sept.) 19

Acute laryngotracheobronchitis occurs most frequen in young children during the seasonal respiratory e demics. It is first characterized by laryngitis w moderate fever, dry cough, subglottic edema and par laryngeal obstruction. As it progresses, tracheobro chial inflammation and edema develop, with exudati of thick, tenacious secretions. As laryngeal obstructi progresses, the cough lessens, sometimes disappear entirely, the temperature drops, sometimes to subnorm and "pale cyanosis" develops as a result of suboxidati and cardiac and circulatory exhaustion from prolong violent respiratory effort (respiratory shock). If t laryngeal respiratory difficulty is relieved by tim surgical intervention, symptoms improve and the te perature rises rapidly because of renewed oxygenati but edema and exudate are still present in the trac and bronchi. If this goes on to obstruction, death occ from exhaustion, obstructive atelectasis or pneumo or all three. If there is bronchoscopic removal of t secretions, there is again improvement, but the infla matory edema persists, and bronchoscopy must be d repeatedly. In spite of this the patient may d Toxemia seems to play a secondary role but is oft

factor, especially in those cases which terminate in eumonia. The mortality rate is extremely high, as own by the reports quoted from numerous authorities.

Baum feels that nonsurgical treatment gives the most pe and discusses his method. He uses convalescent man serum, which may often be secured from some er member of the family from whom the child ac- ired the basic infection. From 50 to 200 cc., depend- on the size of the patient, is given, preferably, ravenously. However, it may be given subcutane- sly or intramuscularly. This procedure may be peated if desired. If this type of serum is not avail- le, then pooled human influenzal convalescent serum ould be used. If the latter cannot be secured, or if adjunct is needed in severe cases, human antistrep- coccic (scarlet fever) convalescent serum should be ed.

Humidified oxygen is exceedingly valuable for reliev- respiratory distress and gives the necessary respite hile other measures are in preparation or taking effect. Hypertonic human plasma may give prompt relief the subglottic and tracheal edema. It comes in the y form and, instead of restoring it to its full volume, e adds only one fourth of the normal volume of ater, which gives a mixture containing four times e normal proportion of plasma protein. This mixture n be given intravenously with perfect safety. By motic action it withdraws the fluid from the edema- us tissues into the blood stream, relieving the obstruc- n. An initial dose of 25 to 40 cc. is usually sufficient; may be repeated as often as necessary. If this is to given, particularly after recent administration of con- lescent serum or in the presence of suspected pul- onary stasis or evident circulatory embarrassment, nesection is advisable, with letting of blood to about ur times the volume of the concentrated plasma to be dministered.

Surgical intervention for the relief of dyspnea, espe- ally tracheotomy, frequently has serious consequences, redisposes to difficulties in the lower respiratory tract nd is to be avoided if prompt and adequate relief can obtained without it. Surgical methods must, how- er, be resorted to without delay in certain cases when is seen that nonsurgical treatment is not securing e desired results. M. V. MILLER, Philadelphia.

Miscellaneous

TUDIES ON CONTROL OF INFECTIONS OF THE RESPIRATORY TRACT (ORAL ADMINISTRATION OF SULFADIAZINE AT THE ONSET OF ACUTE RESPIRATORY ILLNESSES). MORRIS SIEGEL, Am. J. Dis. Child. **66**:114 (Aug.) 1943.

Working in the division of infectious diseases in the ublic Health Research Institute of the City of New ork and having access to children at Letchworth illage, N. Y., and elsewhere, Siegel states that the verity of many acute infections of the respiratory act could be modified if sulfadiazine were used early. ever could be reduced, also the tendency toward pneu- onia or toxemia and the "degree of debility." Be- ween Dec. 14, 1941 and May 2, 1942, 82 treated patients nd 80 controls were observed. The illnesses in the eated were milder and of shorter duration than those the untreated patients. Certain infections, however, peared to be unaffected. In 2 patients lobar pneu- onia was detected on the third day, and 1 of these ed, in spite of sulfadiazine. Withdrawal of the drug some instances may have been too soon, or there may have been undertreatment or a new infection may have set up. The investigation is still in the experimental stage, but it is hoped that more will be found out as to toxicity, sensitivity and strain resistance. Hemolytic streptococci and many types of pneumococci were isolated from smears of secretions from the throats. In about 20 per cent of the cases no significant organism was obtained from the nasopharynx. In spite of the type of onset, the progress of the infection usually appeared to be checked after the first day's treatment, with regression on the second day, which continued to recovery. There were no serious toxic effects from the drug in the dosage used: from 0.5 to 1 Gm. at four hour intervals to keep the blood level of sulfadiazine from 4 to 8 mg. per hundred cubic centimeters during maintenance. VOORHEES, New York.

PENICILLIN AND TYROTHRICIN IN OTOLARYNGOLOGY. S. J. CROWE, A. M. FISHER, ARTHUR T. WARD JR. and M. KATHLEEN FOLEY, Ann. Otol., Rhin. & Laryng. **52**:541 (Sept.) 1943.

Commonly the acute infections of the ears and sinuses are due to the pneumococcus or the beta hemolytic streptococcus, but more often than is realized the primary cause is an anaerobe or some other organism resistant to sulfonamide compounds, and the failure of the physician to make routine aerobic and anaerobic cultures explains ineffective treatment. The sulfonamide compounds are of no value against anaerobic streptococci or other organisms resistant to these drugs. Tyrothricin and penicillin are also selective in their action.

Following the acute primary infection, secondary invaders appear, and many are so resistant to sulfonamide compounds, as well as to tyrothricin and penicillin, that there is no reduction in the suppuration. Even though the organisms are known to be sensitive to a sulfonamide drug, there may be little or no improvement; much depends on their accessibility to the action of the drug. In chronic mucosal infections the organisms are so deeply embedded in the tissues that they are not exposed to the action of drugs which do not penetrate the tissues. Sulfonamide powder and tyrothricin are poorly soluble and do not penetrate. Penicillin is perfectly soluble in water, does penetrate the tissues, is a powerful germicide for certain organisms (most strains of staphylococci, streptococci and pneumococci) and is not toxic, but the authors emphasize that it kills only certain bacteria, and only certain strains of the sensitive bacteria, and has no effect on the growth of others. They give the technic for determining the sensitivity of bacteria to penicillin and tyrothricin.

Sulfonamide compounds in the form of powders, tyrothricin, penicillin and other bactericidal preparations are an aid in preventing infection and hastening healing if used at the conclusion of a surgical operation, as the bacteria are then on the surface and easily reached, but one should know whether the organisms present are sensitive to the drug used.

Penicillin is active mainly against gram-positive cocci, gonococci, meningococci, Clostridium welchii and anaerobic streptococci. The purified extract is active in very high dilutions and is not inhibited by pus, exudates, tissue cells or para-aminobenzoic acid. It is nontoxic even in large doses. Its extraction is com-

plicated, and the supply is still too limited for general distribution. The mechanism of action is not definitely known, but the authors have shown that it is bactericidal in the lower dilutions and bacteriostatic in the higher ones. It acts more slowly than tyrothricin but shows definite killing power in two hours and bacteriostatic powers during the two hours before killing power becomes effective.

Tyrothricin is a bacterial substance isolated from peptone cultures of the anaerobic soil bacterium Bacillus brevis of Dubos. From tyrothricin two crystalline components, gramicidin and tyrocidine, have been separated. Gramicidin is bactericidal for gram-positive organisms only. Tyrocidine contributes to the solubility and stability of the active principle gramicidin. The bactericidal activity of tyrothricin is inhibited by phospholipids, which may explain its relative inactivity in the intestinal tract. It is stable in alkaline mediums or in alcoholic solution, but its in vitro activity is rapidly lost at reactions more acid than p_H 5.5. It has a strong hemolytic action which prohibits its being used intravenously and subcutaneously. To be effective it must come in direct contact with the bacteria. It kills gram-positive aerobic and anaerobic bacteria quickly and is not destructive to tissues. It is effective when used to supplement surgical operations, such as those for chronic sinusitis and mastoiditis. It is good to use instead of saline solution for cleansing irrigation during operation.

Tyrothricin must be applied locally, not injected. It is soluble in alcohol but insoluble in water. Its potency is gradually lost by contact with water either in a test tube or in the tissues. The stock solution (2 per cent in 94 per cent alcohol) is kept in rubber-capped bottles, so that the required amount may be withdrawn as desired and a suspension made with distilled or double distilled water. A 1:10,000 or a 1:20,000 suspension is used for postoperative treatments. More concentration may be used at operation, but in the absence of anesthesia it may cause burning, sneezing and other symptoms due to mucosal irritation.

In acute disease of the sinuses, irrigation with tyrothricin is helpful, but not in chronic cases, since in these the bacteria are deeper within the tissues.

In operations on the frontal sinuses, after the operation is completed, the wound and surrounding parts are thoroughly washed with a 1:5,000 suspension of tyrothricin, and the sinus is packed with gauze soaked with the suspension, bringing the one end out of the nose and closing the external wound. There is rarely any edema of the eyelids, and the incision heals readily. After twenty-four hours the pack is removed under anesthesia with vinethene. The cavity has been rendered sterile or so nearly sterile that excessive growth of granulations is prevented, and therefore the nasal opening does not close if it has been well made. The sinus is irrigated daily with a 1:10,000 suspension for ten days, or a 1:20,000 suspension if titration tests show the organism to be very sensitive. Irrigation should be continued at longer intervals for another two weeks. The authors think tyrothricin is preferable to penicillin in operative work, in which it is important to reduce infection and its consequences to a minimum. If the infecting organism is sensitive to both tyrothricin and penicillin, the best results are obtained by using the former at operation and penicillin, diluted with an equal amount of isotonic solution of sodium chloride, for the postoperative irrigations, as it reaches deep levels and kills organisms the other could not touch.

Tyrothricin dilutions are on a milligram percentage basis, while the penicillin dilutions are calculated approximately in terms of Oxford units. (The Oxford unit is the amount of penicillin that will just inhibit the growth of a standard inoculum of staphylococci in 50 cc. of broth.)

Tyrothricin used as drops or in irrigations for chronic infections of the sinuses or the ears is of little value. Often penicillin is more effective, as it penetrates the tissues more deeply.

Three case histories of patients with extensive osteomyelitis of the skull and facial bones are given to emphasize the value of the use of the two drugs in infections due to organisms that are resistant to sulfanilamide compounds. Other informative tables are given.

M. V. MILLER, Philadelphia.

INTERRELATIONSHIP OF UPPER AND LOWER RESPIRATORY INFECTIONS. JOHN G. MCLAURIN, Ann. Otol., Rhin. & Laryng. **52**:589 (Sept.) 1943.

McLaurin discusses the relationship of various pulmonary conditions to infection of the upper respiratory tract and quotes numerous writers on the subject. He comes to four conclusions: 1. From 85 to 100 per cent of all cases of chronic bronchitis, of bilateral bronchiectasis and of peribronchitis, with the exception of those due to specific pulmonary infections, such as tuberculosis and syphilis, and those resulting from pneumonia, pulmonary abscesses or foreign bodies, are due to infection in the upper respiratory tract, which is nearly always found in the sinuses. 2. The modes of infection have been proved to be (*a*) by descent of droplets or by aspiration, (*b*) by lymphohematogenous extension, (*c*) by hematogenous extension and (*d*) possibly by direct continuity of tissue. 3. Practically all cases of unilateral bronchiectasis not resulting from the presence of foreign bodies, tumors or specific lesions are preceded by atelectasis occurring in infancy or childhood and can be most frequently diagnosed roentgenographically by the demonstration of a rather typical triangular shadow, usually found in one of the lower lobes. He also believes that there are a few cases of bilateral bronchiectasis that can be explained pathologically by atelectasis in childhood. 4. None of the pathologic conditions of the lower respiratory tract mentioned will respond permanently to any type of treatment unless the infection of the upper respiratory tract is successfully taken care of.

M. V. MILLER, Philadelphia.

SOME ASPECTS OF EMERGENCY PLASTIC SURGERY IN WAR INJURIES OF THE FACE. JOHN MARQUIS CONVERSE, Ann. Otol., Rhin. & Laryng. **52**:6[illegible] (Sept.) 1943.

Converse briefly mentions the various types of facial injury encountered and how they are incurred. The aim of the surgeon in treating these injuries is twofold: the prevention and treatment of deformity and the restoration of function. Perhaps because of habituation to bacteria but principally because of an abundant blood supply, facial wounds tolerate well contamination by saliva or by nasal secretion. Compound wounds are drained internally by the cavities, the sinuses and the pharynx. Because of the extensive blood supply, gas gangrene is never seen in the face and deep suppuration

cavernous sinus thrombosis is extremely rare even cases of the most severe facial trauma. Owing to se favorable factors, débridement of facial wounds uld be sparing and not as radical as in wounds elsere. Fragments of fractured facial bones should be served unless they are completely detached.

uccessful treatment requires: 1. Avoidance of tension wounds. If closure of the wound, cannot be accomhed without tension, local plastic flaps must be de; if this is impossible, a primary graft of skin uld be done. 2. Raw areas must be eliminated. This ccomplished by early suture of the wound; if this not feasible, early graft of skin is done. When re is destruction of tissue and penetration into ities lined with mucous membrane, the intervening areas are eliminated by suturing the mucous memne to the skin. 3. The anatomic position of the ues must be preserved. Displaced tissues are replaced their original positions. It is particularly important t when extensive destruction of tissue has taken ce the tissues surrounding the defect be prevented m closing in to obliterate the defect; so edges of n are sutured to mucosal edges. Furthermore, it is ential to fill the defect with a mold supported by extension from a dental splint. This makes subsent repair easier than if a collapse of the tissues had en place with distortion of the angle of the mouth, base of the ala of the nose and the lower lid.

mmediate suturing of wounds of the face is essential. stic operation is much easier at the early stage n it is months later. The wound must be cleansed ore sutures are placed, all foreign material being oved by washing with soap or even by scrubbing r picking out the larger pieces. Débridement must very sparing, and it should be just sufficient to create ncut edges for the wound. In some cases immediate uring is impossible, and the advantage of closure of wound is such that an attempt should be made at ayed suturing. In the interim, if possible, the wound acked with petrolatum-impregnated gauze containing fanilamide.

n delayed suturing of a wound, after cleansing, fanilamide powder is insufflated into all of the und, and the wound is completely closed without inage, or it can be closed with a gauze drain impgnated with sulfanilamide and petrolatum laid in the und with an end coming out of each end of the und, and the latter closed with interrupted sutures. ter six to twelve days the gauze is pulled out gradu- or all at once.

n treating war burns of the face it has been found t the tanning treatment can be used only when the unded persons are seen early. They are thoroughly nsed and then tanned. These agents are uncomtable about the mouth, where the tan tends to crack, l are dangerous around the eyes, where the stiff tan ses exposure of the eyeballs. Treatment of third ree burns is based on early grafting of skin, and tanning agents cause a firm coagulum which retards fting.

The most practical form of treatment of facial burns to cover with an ointment containing a sulfonamide mpound after cleansing. Trichloroethylene will dis- ve the oil which often covers burns occurring on gs. Usually in third degree burns the extent of truction can be determined and this area excised, d a split skin graft applied. When the eyelids are involved, early grafting of skin is essential to prevent ectropion.

Compound facial injuries with cerebrospinal rhinorrhea require careful evaluation by the use of roentgenograms and laminographs. Air in the ventricles shows communication with the sinuses and demands operation, as does extensive comminution in the cribriform area suggesting penetration of bony fragments into the brain.

Comminution of the malar bones demands careful roentgen study to determine whether correction should be done by the Caldwell-Luc approach or by incisions made over the site of fracture. It is necessary to reconstruct the floor of the orbit to prevent diplopia.

M. V. MILLER; Philadelphia.

THE ETIOLOGY AND THERAPY OF SPASTIC SPEECH. MAURICE H. KROUT, J. Speech Disorders **7**:193 (Sept.) 1942.

Some of the literature of stuttering regarding incidence, theories of causation and symptomatology is surveyed, and some of the methods for treatment are presented. No method is completely successful. The cause of stuttering is not fully known.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

A STUDY OF THE ONSET AND DEVELOPMENT OF STUTTERING. WENDELL JOHNSON, J. Speech Disorders **7**:251 (Sept.) 1942.

Forty-six stuttering children were compared with 46 nonstuttering children in regard to birth conditions, general developmental conditions, speech development, diseases and injuries, stuttering, handedness in the family background and handedness and eyedness of the group studied. No significant facts were disclosed. Johnson suggests that such stuttering in its serious forms develops only after a diagnosis of stuttering is made and as a consequence of the diagnosis.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

A STUDY OF THE INFLUENCE OF COMMUNICATIVE RESPONSIBILITY IN A CHORAL SPEECH SITUATION FOR STUTTERERS. JON EISENSON and CHARLOTTE WELLS, J. Speech Disorders **7**:259 (Sept.) 1942.

In a study of 19 stutterers, 9 stutterers read aloud with an individual clinician and then read a similar passage to a hypothetic audience. There apparently was some decrease in the quality of their speech when an actual communication to an audience was requested, but the results were for the most part not mathematically significant.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

INTRACRANIAL ARTERIOGRAPHY WITH RAPIDLY EXCRETED IODINE COMPOUND (DIODRAST). J. LAWRENCE POOL and STEWART ALEXANDER, New York State J. Med. **43**:1429 (Aug. 1) 1943.

The authors state that intracranial arteriography is a useful diagnostic procedure which has fallen into disrepute because the dye (thorium dioxide) is radioactive. Diodrast, however, is not radioactive and is completely out of the body in a few hours after intravenous or intra-arterial injection. Since July 1939 a 35 per cent solution has been used in Bellevue Hospital on 9 patients. In every patient one could see by roentgenogram the outline of blood vessels within the skull on

the side on which the vessels had been injected. The internal carotid artery is used, but it is necessary to give a preliminary hypodermic injection of morphine sulfate, ⅙ to ¼ grain (0.011 to 0.016 Gm.), with scopolamine hydrobromide, 1/150 grain (0.43 mg.). The skin is infiltrated with a 1 per cent solution of procaine hydrochloride. A 3 cm. incision is made parallel to the border of the sternocleidomastoid muscle upward from the level of the thyroid gland. The external carotid artery is exposed and retracted by a coarse ligature so that the internal carotid artery is brought to view. This artery is similarly drawn aside. Then the carriage is trundled to the x-ray room, a no. 18 needle is inserted into the internal carotid artery and diodrast is inserted. There are other details to the technic which must be studied to be understood. The claim is made that this preparation in 35 per cent solution provides a ra paque medium for intracranial arteriography. Stro solutions are not safe. With the patient under l anesthesia, from 8 to 15 cc. may be injected as m as four times at one sitting without ill effects.

VOORHEES, New Yor

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. Ma COHN, Rev. de otorrinolaring. **2**:102 (Dec.) 194

Cohn presents a case of acute pharyngitis which veloped in a girl 3½ years old and became complic by thrombosis of the cavernous sinus in two days. received intensive treatment with sulfapyridine and quent small blood transfusions. She made a com recovery.

PERSKY, Philadelphi

News and Comment

COND SOUTH AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY

The second South American Congress of Otorhinolaryngology will be held in Montevideo, Uruguay, ing the first half of October. Papers on the following subjects will be presented:

Prophylaxis of Deafness, by Prof. Eliseo V. Segura and Prof. Eduardo Casterán, Argentina.

The Post-Operative Care of Mastoiditis, by Prof. Felix Veintemillas, Bolivia.

Bronchoscopy in Diagnosis and Treatment, by Dr. Plinio de Mattos Barretto, Brazil.

The Importance of Examination of the Eighth Nerve in the Tumors of the Brain, by Dr. Riesco M. C., Chile.

Leishmaniasis in Otorhinolaryngology, by Crispín Insaurralde, Paraguay.

Rhino-Scleroma, by Prof. Juvenal Denegri, Peru.

Cancer of the Larynx, by Prof. Justo M. Alonso, Uruguay.

Further information may be secured by writing to the General Secretary of the Executive Committee, Dr. Juan Carlos Oreggia, Yi 1491, Montevideo, Uruguay.

EXAMINATION IN OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct an examination in Chicago, at the Palmer House, on Oct. 4, 5, 6, and 7, 1944.

Book Reviews

aumatic Injuries of the Facial Bones. By John B. Erich, M.S., D.D.S., M.D., consultant in laryngology, oral and plastic surgery at the Mayo Clinic, and Louie T. Austin, D.D.S., head of section on dental surgery at the Mayo Clinic. In collaboration with the Bureau of Medicine and Surgery of the United States Navy. Pp. 600, with 333 illustrations. Price $6. Philadelphia: W. B. Saunders Company, 1944.

The introduction is by Surgeon General McIntire of e United States Navy. He is "particularly impressed" the manner of presentation of this subject, which is important to the armed forces at this time. This anual tells how to do the "immediate thing" quickly, rrectly and well. The "ordinary practitioner" will nfit by it as well as the beginner in plastic surgery r injuries due to trauma. In another foreword Dr. nald C. Balfour, of the Mayo Clinic, says that the inciples of plastic surgery are now well established in neral and that those concerned need only to apply ese principles to special problems which arise in conction with casualties to face and jaws. Great Britain was the first to set up and develop special clinics for plastic surgery during World War I, and the young men of the United States learned much from Mr. Gillies and others who were generous enough to share their knowledge and work. In brief, the subject matter of this book is based on requests from members of the armed forces who have taken special training at the Mayo Foundation. No one can go far in this kind of surgery without a knowledge of prosthetic dentistry. The appliances invented and used by dentists are often quite intricate in their manufacture and fitting, but "restorations" often make the difference between a good and a poor end result. This is especially true of fractures and loss of the mandible for which bone grafting, wiring of fragments and the like are procedures which require both skill and patience, plus *time*.

Each problem is illustrated on the left hand page in the book. This is merely an illustration of the lines of fracture. On the right page, the text describes what must be done to secure a satisfactory result. This is a useful and practical book. One wonders, however, whether the title is not tautologic? Perhaps this is mere quibbling.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

Place: Chicago. Time: June 12-16, 1944.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.

President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.

Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

Place: Palmer House, Chicago. Time: Oct. 8-11, 1944.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.

Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

Place: New York. Time: June 6, 1944.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

Place: New York. Time: June 7-8, 1944.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

Place: New York. Time: June 9-10, 1944.

SECTIONS:

Eastern.—Vice President: Dr. Westley M. Hunt, 907 5th Ave., New York 21.

Southern.—Vice President: Dr. William C. Warren Jr., 478 Peachtree St. N. E., Atlanta, Ga.

Middle.—Vice President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.

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American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York 21.

Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

Place: New York. Time: June 5-6, 1944.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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